The Australian and New Zealand
JOURNAL OF SURGERY

Vol. 29 — No. 3 FEBRUARY, 1960

FLUID AND ELECTROLYTE BALANCE IN SURGERY*

By Eric M. Nanson

Department of Surgery, University of Saskatchewan, Saskatoon, Canada

THE last twenty-five years have produced a remarkable advance in the understanding of the body's fluid and electrolyte needs and their regulation. Particularly is this so in the field of surgery. During this period there have been four phases in the post-operative care of the water and salt needs of the surgical patient. The first of these was the period of inadequate supply, i.e. dehydration. The second was the period of incautious use of both water and salt causing saline overload. The third was the period of failure of understanding of the different types of the electrolyte deficit picture, and the fact that different combinations of electrolytes are required to correct different situations. The last and present period is that of appreciation of the importance of potassium in electrolyte replacement.

In any fluid balance problem, the water requirements and the electrolyte requirements have to be considered together but for descriptive purposes it is easier to discuss them separately. Therefore, the fluid problem will be dealt with first. It is important to recall to mind the three fluid compartments of the body, viz. intracellular, interstitial and intravascular compartments. The relative distribution between these three compartments varies according to the age of the person; furthermore, the exact distribution is not agreed upon by all writers, but Fig. I gives average and acceptable proportions. The intracellular fluid is the body water vital for normel cell function and must remain relatively constant unless electrolyte shift occurs as well, because the osmolarity of the cell is a relatively constant factor. The interstitial fluid bathes the cells and so acts as a reservoir and buffer to protect the cells against undue fluctuation in water content of the body. It is the most labile of the fluid compartments from the point of view of volume and composition. The intravascular compartment can be looked upon as the conveyor belt which distributes food, electrolytes and fluid round the body. Essentially the gains and losses from the body are mediated through this intravascular compartment. This is illustrated in Fig. II which has been adapted from Gamble.

Fluid may move freely from one compartment to another. The force which causes this movement is the osmotic pressure of the electrolytes and proteins in the respective compartments. Because of its contained proteins, the intravascular compartment tends to maintain its volume longer than the interstitial compartment. This is of course necessary to maintain a circulating blood volume. When this balance is destroyed and the intravascular blood volume falls to a certain level, peripheral vascular collapse occurs. If this situation is uncorrected or progresses, death results from oligemic shock. This is the type of death caused by gross electrolyte and fluid deficit.

In general, surgery deals with fluid and electrolyte deficits. Therefore, it is important to consider the normal intake and output of fluid over twenty-four hours, under ordinary temperate climatic conditions. This is set

*Substance of a lecture delivered in Auckland, August, 1958.

out in Fig. III. The balance has to be regulated both on the intake and output side.

The fluid intake is regulated by:

(1) thirst,
(2) appetite,
(3) desire,
(4) oxidation of food, producing water of oxidation.

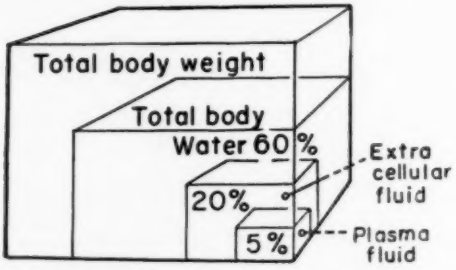


FIG. I. Relative distribution of body water in the adult.

The prime regulator is thirst, which is mediated through the hypothalamus. Here, very sensitive osmoreceptors are situated which initiate the sensation of thirst and this prompts fluid intake. Appetite, to a lesser degree, rules the intake insofar as it causes a certain amount of water to be taken in with food, and also, of course, regulates the amount of oxidation water produced. But appetite is to a large extent influenced by habit and hand in glove with this goes desire which is the result of habit. This may, to a certain minor degree, amount to addiction in some cases. A person may be in the habit of drinking tea or coffee, or beer or whisky, so many times a day, but need plays little part in this. Thirst is the great stimulator to adequate fluid intake. However, if electrolytes in excess of fluid are lost, the hyperosmolarity in the hypothalamus will be absent. Under these circumstances thirst will be absent even though there is a gross fluid deficit, because the electrolyte deficit is the greater.

The normal output of fluid from the body is regulated by (see Fig. IV):

(1) the anti-diuretic hormone,
(2) the heat regulating centre,
(3) the absorption of fluid from the large bowel,
(4) the secretion of fluid from the respiratory tract.

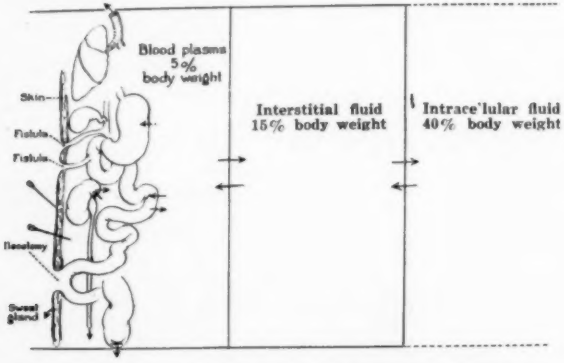


FIG. II. Diagram to show the adding and removing of fluid from the vascular compartment.

AVERAGE NATURAL INTAKE AND OUTPUT OF WATER

Intake		Output	
Drink	1,500 cc.	Urine	1,500 cc.
Solid food	850 cc.	Lungs	400 cc.
Oxidation of food	250 cc.	Skin	600 cc.
		Faeces	100 cc.
	2,600 cc.		2,600 cc.

FIG. III. Normal fluid balance in 24 hours.

The anti-diuretic hormone is secreted by the posterior lobe of the pituitary gland under the stimulus from the osmoreceptors of the hypothalamus. If there is excess fluid in the body, then the osmoreceptors will be stimulated to appreciate a hypo-osmolarity and therefore they will cut down the stimulus to the posterior lobe of the pituitary which will consequently secrete less anti-diuretic hormone. Therefore, there will be a "slacker rein" kept on the kidney. There will be less reabsorption by the renal tubules, and therefore, more urine will be passed and the over-dilution of the electrolytes of the body will be corrected. In the case of dehydration the whole process is reversed by the increased activity of the osmoreceptors. These cause the posterior pituitary to secrete more anti-diuretic hormone which cuts down the amount of urine secreted.

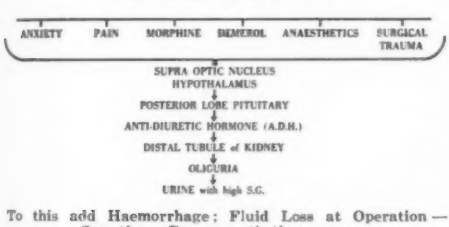


FIG. IV The regulation of urinary output by antidiuretic hormone.

The heat regulating centre plays its part by regulating the amount of sweat secretion and consequently is only involved incidental to heat regulation. The control of the amount of absorption of fluid by the large bowel is not clearly understood, but a dehydrated person will be constipated and therefore has marked fluid absorption in the colon and rectum. This may be secondary to stasis. On the other hand, if a bowel disease exists causing diarrhoea, excessive fluid is lost by this route because the large bowel has not got time to absorb as much water as it should.

The fluid lost from the lungs is usually a fixed amount varying from 500-800 cc. daily. It is dependent on the humidity of the atmosphere and has no regulating mechanism such as the hypothalamus-pituitary axis. It is in essence a fixed amount for a given atmospheric state.

The regulation of the daily balance may be upset by:

(1) deficient intake,

(2) excessive loss,

(3) the effects of trauma, stress, haemorrhage or surgery on the regulating mechanism.

(1) *Deficient intake*

This may be due to many conditions. Thus, nausea and vomiting, such as occur in an intestinal obstruction or a pyloric stenosis, will prevent adequate intake and absorption. A carcinoma of the oesophagus will prevent swallowing. After a head injury, or in cases of coma from any cause such as an intracerebral tumour, intake will be nil. Such a situation has to be overcome either by parenteral feeding or by gavage.

(2) *Excess loss*

Excessive loss may occur in a large number of ways, but there are two major divisions of these ways:

viz. (1) overt loss,
(2) hidden loss.

Overt loss

May occur by:

(1) vomiting,

(2) diarrhoea,

(3) sweating,

(4) fistulae—gastric, duodenal, jejunal, ileostomy, biliary and pancreatic.

It is often not realized the quantities of fluid that can be lost by these means. Therefore, it is convenient to refer to Marriott's Chart (Fig. V) which depicts the maximum loss possible in twenty-four hours by the various routes. It will be noted that vomiting can produce loss of 6,000 cc. daily and diarrhoea 7,000 cc. daily.

It is often difficult to know how much fluid loss to allow for from sweating. Once again a convenient table is provided by Marriott (Fig. VI). It will be seen that marked sweating with wetting of the bedding will cause a loss of upwards of 2 litres of water per day.

Hidden loss

In hidden loss, the fluid has not actually left the body, but it has become pooled and stagnant. It is no longer of any use to the

economy of the body. It is, so to speak, temporarily out of circulation. If it can be made to return to active circulation, then its usefulness is restored. This type of situation occurs with burns. In the burnt area a marked amount of oedema fluid is poured out into the tissues, quite apart from that which is lost, in the form of serum, from the burnt surface. A similar situation exists in crush injuries where blood and oedema fluid is lost into the traumatized area. The same applies in fractures. It has been calculated that in a fractured shaft of femur, upwards of 1,500 cc. of fluid may be lost into the fracture site. When a major vein, such as the common iliac vein, is suddenly obstructed by deep venous thrombosis, a large amount of useless oedema fluid is trapped in that leg as evidenced by the gross swelling. When bowel ileus occurs, whether it be dynamic or adynamic, vast quantities of fluid are poured out into the dilated bowel; this fluid is thus temporarily lost to the circulating fluid of the body. A similar situation occurs with the rapid onset of ascites, or worse still, in the rapid re-accumulation of ascites after an extensive volume has been removed by paracentesis or operation. Such rapid re-accumulation may precipitate an acute water and electrolyte deficit with the dire results of circulatory collapse. The same may be said of a marked peritonitis in which fluid will quickly accumulate in the peritoneal cavity.

Assessment of fluid deficit

Having decided how the fluid is lost, the next important procedure is to try and estimate how much fluid has been lost, and therefore, how much must be given back. The ideal method would be to know the exact amount of weight lost by the patient as a result of the fluid loss; but unfortunately, it is rare for the correct weight to be known just before the patent began to lose water and therefore the baseline is so often missing. Hence empirical methods have to be used. The use of laboratory methods such as the estimation of blood volume and the extracellular fluid space are, by and large, rarely applicable. They do not give a true estimate of the amount of intracellular fluid lost.

PARENTERAL FLUIDS IN SURGICAL PATIENTS

Plan for Determining Sensible and Insensible Losses of Water and Chloride

Degree of perspiration	*24-hour water and chloride loss*	
	Water (ml.)	*Chloride (mEq.)*
Absence of fever and perspiration	1,000	0
Obvious perspiration and fever	1,500-1,800	40
Marked perspiration requiring change of bed linen and pyjamas	2,000 2,500	80

FIG. VI. (After H. L. Marriott.) Possible volumes of fluid loss by sweating.

Once again Marriott has provided a useful chart for such an empirical estimate (Fig. VII). It should be pointed out that, in obtaining a history from the patient, a rough estimate of the amount lost can sometimes be

POSSIBLE SECRETION LOSSES (24 HOURS)

(From H. L. Marriott: Water and Salt Depletion.
Courtesy Charles C. Thomas, Publisher, Springfield, Illinois.)

Secretion	*Circumstances of abnormal loss*	*Volume (ml.)*	*NaCl Equivalence*	
			NaCl (gm.)	*= Isotonic Saline (Litres)*
Sweat	Sweating	14,000	35	4
Saliva	Vomiting or	1,500	8	1
Gastric juice	Gastric suction	2,500	18	2
Bile	Fistula	500	4	½
Pancreatic juice	Fistula	700	6	¾
Intestinal juices	Diarrhea; Intestinal suction	3,000	22	2½
Mixed secretions:				
Vomit	Vomiting or suction	6,000	40	4½
Diarrhea	Diarrhea	7,000	50	6

FIG. V. Chart to show possible volumes of fluid loss from the body by various routes.

made by finding approximately how much material has been vomited, or lost by diarrhoea.

If, therefore, a patient is moderately severely dehydrated, according to Marriot, he would have lost 6 per cent. of his body weight as fluid, which in a 70 kilogram man would be 4,200 cc. Therefore, the deficit would be 4,200 cc. The basal needs would be approximately 2,600 cc. If further loss is to be expected in the next twenty-four hours, say 1,000 cc., this must be allowed for. Therefore, this patient would require 4,200 cc. + 2,600 cc. + 1,000 cc. = 7,800 cc. of water in the next twenty-four hours.

STAGES OF WATER DEPLETION

(Marriott) — 70 kilo man

1. Early:	
Thirst	2 per cent. body wt.
	1,400 cc.
2. Moderately Severe:	
Marked thirst	6 per cent. body wt.
Dry mouth	4,200 cc.
Oliguria	
Weakness	
Malaise	
Loss of skin turgor	
Low eyeball tension	
Some mental changes	
3. Very Severe:	
As above	7-14 per cent. body wt.
Severe mental changes	5,000
Hallucinations	10,000 cc.
Gross weakness	

FIG. VII. Empirical estimate of degree of fluid depletion.

Methods of replacement of fluid

By far the best method of replacing lost water is by mouth if that is feasible. This is often so in the less severe burns. The gut can then accept or reject the quantities that the body needs. In patients who are unconscious, this route may still be used by means of a Levine tube. Unfortunately, however, so many of the patients whom the surgeon is called upon to treat are unsuitable for this route of replacement because the absorptive mechanism is upset, as is the case in intestinal obstruction. Therefore, the parenteral route has to be used. Gastrostomy and jejunostomy may occasionally have a place but by and large, they are rarely used. Replacement of fluid by means of a rectal drip has unfortunately lost its popularity. The rectal route has a very definite place, and it should be used more frequently. Provided only water and not electrolytes is to be given and provided that not more than 100 cc. per hour are required, it is a useful route. Ordinary tap water may be used. No sterile precautions are required. It can be set up and replaced by a nurse. The water may be quietly absorbed while the patient sleeps. As long as not more than 100 cc. per hour are given and as long as this is run in, in the form of a drip using a No. 14 urethral catheter, patients will usually tolerate the procedure well. It is, of course, not suitable in large bowel surgery.

The subcutaneous route is still useful in babies, and now that hyaluronidase is available to aid absorption, it has a real place. It should be pointed out that only normal saline or some isotonic solution should be used.

But, by and large, the most widely used and applicable route is the intravenous one.

Assessment of the success of the fluid replacement

The success of the treatment employed should be gauged by the clinical improvement of the patient. Thirst should regress, the tongue should become moist, skin should regain its elasticity and the muscles their tone. The sensorium should begin to clear. Any patient in severe fluid and electrolyte deficit should have an indwelling Foley type catheter inserted into the bladder, and the hourly output of urine should be recorded. When success attends the doctor's efforts, the urinary output should rise hour by hour until the optimum quantity of 50 cc. per hour is obtained. A severe electrolyte and fluid problem requires the frequent observation of the attending doctor, and assessment should be made every four hours and adjustments to therapy made according to the response obtained.

Electrolytes

Up to the present, only fluid replacement has been considered. The electrolyte problem will now be dealt with. This is a convenient method of discussing the subject but of

course, from a practical point of view, fluid and electrolytes are inextricably bound up together and one cannot be considered without the other, except on the theoretical basis.

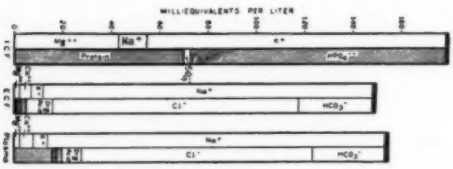


FIG. VIII. Normal electrolyte content of the three fluid compartments of the body.

In considering electrolytes, it is important to have a clear picture of their normal distribution between the compartments as is set forth in Fig. VIII. It will be noted that to all intents and purposes the interstitial fluid and the intravascular fluid are similar in electrolyte composition. On the other hand, the intracellular fluid is vastly different in composition. The main anion is potassium and the main cation is phosphate. At one time it was thought that there was little interchange of K and Na across the cell membrane. This is now known to be untrue. The important ions to be considered in all this work are potassium (K^+), sodium (Na^+) and chloride (Cl^-).

The normal route of intake of electrolytes is by mouth. The normal output is by the urine, the faeces and the sweat. The main normal regulator of the electrolyte levels is the kidney. The normal functioning of the kidneys is therefore of vital importance to normal electrolyte control. Therefore, in any electrolyte deficit problem, it is essential that adequate urinary excretion be restored and maintained as soon as possible. The scheme of this kidney regulation is set out in Fig. IX. By means of the hypothalamus-pituitary-adrenal axis, the excretion of Na and K is regulated. This normal regulation may be upset by the same four ways that fluid regulation is upset:

(1) deficient intake,
(2) excess loss,
(3) trauma and stress,
(4) renal disease.

Deficient intake requires no further comment.

Excess loss

This may be:

(1) overt, or
(2) hidden,

as was the case with fluid loss.

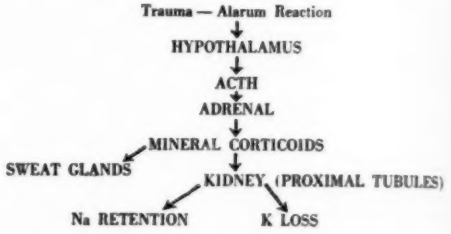


FIG. IX. Scheme of kidney regulation of electrolytes.

The volumes of electrolyte carrying fluid which can be lost by overt means should be recalled by referring back to Fig. V. But it is important to know the composition of these various types of fluid with respect to Na^+, Cl^- and K^+. This varying composition is set out in Figs. X and XI in which a comparison is made with tissue fluid.

As regards hidden fluid loss, such as oedema fluid, fluid lost into the bowel by ileus or ascitic fluid, the composition of the fluid may be regarded as a transudate of the blood; in other words, its composition may be regarded as the same as that of tissue fluid.

Trauma

Trauma and stress, such as occurs from operation, from accident and from disease, works through the alarum reaction of Selye. This is well set out in Fig. IX. The stress stimulates the hypothalamus, which stimulates the pituitary, which secretes adrenotropic hormone, which stimulates the adrenal cortex to secrete more aldosterone, which acts of the proximal renal tubules to cause Na^+ retention and K^+ loss. An extra amount of glucocorticoids is also secreted which will increase protein catabolism and hence a loss of K^+.

Electrolyte deficit

By and large, surgery is usually concerned with electrolyte deficits. It is therefore important to be able to recognize the signs and symptoms of electrolyte deficit. This has

been well set out by Marriott in Fig. XII. Here pure water deficit is compared with pure salt deficit. In actual practice, a pure deficit of either water or electrolyte practically never occurs. There is nearly always a mixture of the two. In water depletion there is a state of hyperosmolarity. In salt depletion there is a state of hypo-osmolarity. In this state of hypo-osmolarity three major effects are seen:

There is:

(1) oedema of cells and relative excess of intracellular fluid which, in the case of the brain, produces gross disorientation,

(2) loss of circulating blood volume resulting, ultimately, in peripheral circulatory collapse, and

(3) an upset in the normal functions of the enzymatic systems of the cells.

Calculation of electrolyte deficit

There are two methods of calculating the electrolyte deficit and consequently the amount required to restore the balance:

(1) by empirical methods (Marriott).

(2) by the blood analyses.

The empirical assessment method has been set out by Marriott and is shown in Fig. XIII. In these charts Marriott has stated the deficit as grams of NaCl. This presupposes that an equal quantity of Na and Cl is lost. This may or may not be so. Usually it is not so. However, the important ion to consider is the anion Na. Therefore the deficit is expressed as milliequivalents (mEq) of Na. Having therefore decided approximately how much Na ion is required, the amount of Cl ion required expressed as mEq can be calculated with reference to the type of fluid that has been lost. This may be done by deciding whether there is overt loss or hidden loss or a combination of the two. In hidden loss, the fluid lost usually has the composition of a tissue fluid, i.e. Na will be roughly 140 mEq/litre and Cl will be 100 mEq/litre. Therefore, in selecting a repair solution, one which has this type of composition should be used — e.g. Ringer's lactate, which contains 131 mEq of Na and 107 mEq of Cl.

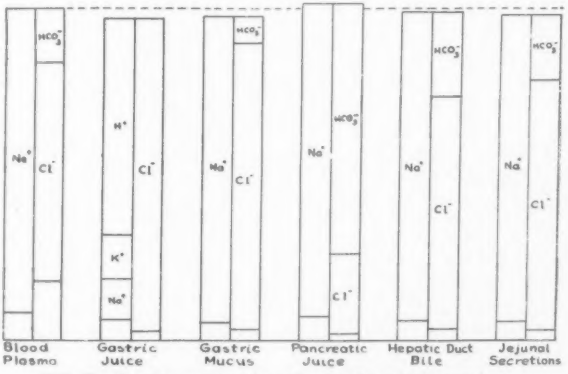


FIG. X. Varying composition of gastro-intestinal secretions.

In overt loss, as in vomiting or diarrhoea, the composition of the fluid has to be assessed by referring back to Figs. X and XI. Profuse diarrhoea and the profuse vomiting of intestinal obstruction causes a loss of fluid and electrolyte such as is found in a transudate of the blood, i.e. Na/Cl = 4/3. Consequently, again a solution such as Ringer's lactate should be used. In the vomiting of pyloric stenosis much more Cl than Na will be lost but K will also be lost. This is always contingent on the patient not being achlorhydric, either temporarily or permanently. In this case, a solution, containing more Cl than Na relative to tissue fluid, should be used. In general, this will be physiological saline which contains 154 mEq of Na and 154 mEq of Cl relative to 140 mEq of Na and 100 mEq of Cl in blood plasma. In severe cases of alkalosis it is permissible to use ammonium chloride (NH_4Cl) solution at a concentration of 165 mEq of Cl per litre. In the event of a pancreatic fistulae it is important to regard this loss as pure Na+ and therefore replace this fluid lost, volume for volume, with M/6 sodium lactate which contains 167 mEq/litre of Na and no chloride.

In calculating electrolyte replacement of a patient who is continuing to lose electrolytes from gastric aspiration, from fistulae or diarrhoea, an extremely useful method is that of Scribner, of the Mayo Clinic. In this method a large bucket is kept in the patient's

room and the whole of the fluid lost for twenty-four hours is kept in this bucket. All gastric aspiration, vomitus, diarrhoea, urine and fistulous fluid, is emptied into this bucket. At the end of the twenty-four hours the volume of this fluid is measured. A sample of it is sent to the laboratory for estimation of the content of Na, K and Cl expressed as mEq/litre and hence, knowing the volume, the exact amount of these ions lost in the past twenty-four hours is known and can be replaced in the next twenty-four hours.

work. The values that are essential are serum Na, serum K, serum Cl and serum bicarbonate. It is advantageous to have the serum pH, if this is feasible.

Using the values of the serum electrolyte levels, the deficits of Na and Cl may be calculated as shown by Fig. XIV. It should be noted that Cl deficit is calculated on the extracellular fluid volume only, whereas the Na deficit is calculated on the total body fluid. K is not usually calculated in this

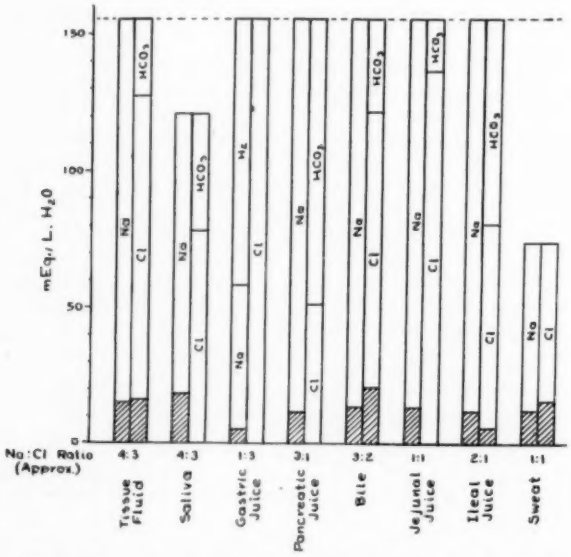


FIG. XI. Varying composition of body secretions as regards anion and cation.

Calculation by the blood analyses

With any patient who is dehydrated and in salt deficiency, it is wise to have the serum electrolytes estimated as soon as possible, and to be guided in the treatment by the values so obtained. Furthermore, in a difficult case it is wise to have these estimates repeated periodically as is necessary. It should, however, be pointed out that serum electrolyte estimations are not essential to the successful treatment of a fluid balance problem, and in many situations where these facilities are not available, intelligent and entirely satisfactory treatment can be carried out to a successful conclusion. The initial electrolyte levels are very valuable as a base line from which to manner and indeed is rarely calculated quantitatively but rather qualitatively. The reason for this will be explained later.

Yet another method of calculating electrolyte deficits, is to take the estimated fluid deficit, e.g. 4,000 cc. in a moderately severely dehydrated 70 kilogram person and then to assess the approximate electrolyte composition of the fluid lost on an empirical basis. Thus with an intestinal obstruction or with severe diarrhoea this could be assumed to be a transudate of the blood and consequently a repair solution such as Ringer's lactate or lactate saline should be used.

COMPARISON OF EFFECTS OF WATER AND SODIUM CHLORIDE DEPLETION

(From H. L. Marriott: Water and Salt Depletion.
Courtesy Charles C. Thomas, Publisher, Springfield, Illinois.)

Manifestation	*Pure water depletion*	*Pure salt depletion*
Dehydration	+++primary or simple	+++secondary or extracellular
Thirst	+++	Absent
Lassitude	+	+++
Orthostatic faintness	Absent till late	+++
Urine volume	Scanty	Normal till late
NaCl in urine	Often+	Always absent except in Addison's disease
Vomiting	Absent	May be +++
Cramps	Absent	May be +++
Plasma NaCl	Slight increase or normal	Diminished +++
Blood urea	+	+++
Plasma volume	Normal till late	Decreased +++
Haemoconcentration	Not till late and slight	+++
Blood viscosity	Normal till late	Increased +++
Blood pressure	Normal till late	Fall +++
Water absorption	Rapid	Slow
Mode of death	? due to rise of osmotic pressure	Peripheral circulatory failure

FIG. XII. Table of symptoms of pure water and pure salt depletion.

EMPIRICAL ESTIMATE OF SALT DEFICIENCY

(70 kilo man)

1. Slight to moderate:
   Urinary chlorides—low or absent
   Lassitude
   Faintness on sitting up
   } 0.5 gm/kilo NaCl
   = 35 gms. NaCl
   = 620 mEq Na.

2. Moderate to severe:
   Absent urinary chlorides
   Lassitude
   Syncope
   Nausea
   Vomiting
   Systolic Blood Pressure below 100 mm/Hg
   } 0.5 gm/kilo
   } 0.75 gm/kilo NaCl
   35 – 52 gms. NaCl
   620 mEq – 900 mEq Na.

3. Very severe:
   Absent urinary chloride
   Apathy
   Stupor
   Nausea
   Vomiting
   Coma
   Systolic B.P. below 90 mm/Hg
   } 0.75 – 1.25 gms/kilo NaCl
   = 52 – 87 gms. NaCl
   900 mEq – 1,500 mEq Na.

Restore by hypertonic NaCl 5 per cent. = 900 mEq/litre approx.

FIG. XIII
(1) Empirical assessments of electrolyte deficit according to H. L. Marriott.
(2) Empirical assessments of electrolyte deficit according to H. L. Marriott.
(3) Empirical assessments of electrolyte deficit according to H. L. Marriott.

In making calculations for the next day's requirements of fluid and electrolytes, it is important to keep abreast of the loss rather than to be catching up from the day before. Thus the fluid and electrolytes given should consist of the deficit up to that day, plus the basal needs of the coming day, plus any expected loss, as by gastric aspiration over the next twenty-four hours. Unless this policy is followed, the intravenous therapy is always twenty-four hours behind what it should be (Fig. XV).

ELECTROLYTE REQUIREMENTS

By Blood Chemistry Calculation

70 kilo man
Na = 130 mEq/litre = Deficit of 10 mEq/litre
Cl = 70 mEq/litre = Deficit of 30 mEq/litre
HCO_3 = 40 mEq/litre = Excess of 13 mEq/litre
K = 5 mEq/litre = Normal

Total Na: Calculated from total body fluid
= 70 x 60 per cent. = 42 litres
is 42 x 10 = 420 mEq deficit

Cl: Calculated from extracellular fluid
= 70 x 20 per cent. = 14.0 litres x 30
= 420 mEq of Cl.

FIG. XIV. Calculation of electrolyte deficits by blood chemistry method.

Repair solutions

It has been the habit in the past to use normal or physiological saline as the standard repair solution without due regard to the

electrolyte need of the patient. In most instances of minor fluid and electrolyte disturbance, this is satisfactory because, although the physician may put into the patient the wrong proportion of electrolytes, yet the patient's kidneys can and do correct the error. However, in severely dehydrated and electrolyte-deficient patients this is not satisfactory, because the kidney function will be depressed and the deficits are large. Here the choosing of the correct type of repair solution is important.

FLUID REQUIREMENTS FOR FIRST TWENTY-FOUR HOURS FOR SEVERELY DEHYDRATED MAN

4,200 cc. = Deficit
3,000 cc. = Basal needs
? 1,000 cc. = Continued expected loss
8,200 cc. Total

Electrolyte requirements — Empirical calculation

For a 70 kilo man — loss from low intestinal obstruction:

Deficit = 4,000cc. of extracellular fluid.

This should contain 4 x 140 = 560 mEq Na.

This amount of Na would be contained in about 4 litres of Ringer's lactate or lactate saline.

But basal needs will require about 100 mEq of Na and it can be expected that about 100 mEq further will be lost over the next twenty-four hours. Therefore, over the next twenty-four hours give:

Deficit	= 4,000 cc.	= 560 mEq Na
Basal needs	= 2,500 cc.	= 100 mEq Na
Expected further loss	1,000 cc.	= 100 mEq Na
	7,500 cc.	760 mEq Na

Order 5 litres of lactate saline
2 litres of glucose and distilled H_2O
This will give 750 mEq of Na
500 mEq of Cl.

FIG. XV. Calculation of fluid requirements over next 24 hours in a moderately dehydrated man.

The repair solutions available and of general and wide use are as follows (Fig. XVI):

Of these repair solutions, the most useful and generally used are normal saline, Ringer's lactate and M/6 lactate. Ammonium chloride is rarely necessary, and then should be given slowly. Generally, where a solution is required to give a relatively larger proportion of Cl as compared with Na, then normal saline is indicated. Because, relative to the extracellular fluid, this solution contains $\frac{1}{3}$ more Cl than does the extracellular fluid.

Ringer's lactate solution is very useful in many surgical conditions such as intestinal obstruction, excessive diarrhoea and paralytic ileus. It should be more widely used. The amount of K and Ca it contains are of no particular value and need not be considered.

M/6 lactate is a very useful solution to bring up a marked Na deficit — it has particular application in diarrhoea such as occurs in mucous membranous colitis.

The hypertonic solutions of Na Cl are useful in severe electrolyte deficits with relatively less dehydration. Again this situation is rarely met in surgical work unless an undue amount of fluid without electrolytes has been given the patient beforehand.

In order to summarize the foregoing discussion, a patient will be considered who is suffering from severe intestinal obstruction and who is in severe electrolyte and fluid deficit. Such a patient should be able to be prepared for operation by vigorous replacement therapy within six hours.

Initially a Levine tube will be put down and naso-gastric suction instituted forthwith. An intravenous drip of Ringer's lactate will be set up and 1,000 cc. should be allowed to

(1) Normal saline = 0.9 per cent.	= Na 154 mEq/L, Cl 154 mEq/L.
(2) Ringer's lactate	= Na 131 mEq/L, Cl 107 mEq/L.
(3) 1/6 Molar lactate	= Na 167 mEq/L, Cl
(4) Lactate saline	= Na 152 mEq/L, Cl 97 mEq/L.
(5) Ammonium chloride soln. 0.85 per cent.	= Na nil Cl 167 mEq/L.
(6) Sodium chloride 2 per cent.	= Na 315 mEq/L, Cl 315 mEq/L.
3 per cent.	= Na 520 mEq/L, Cl 520 mEq/L.
5 per cent.	= Na 860 mEq/L, Cl 860 mEq/L.

run in thirty minutes. This rapid rate is necessary because the patient is extremely dry and can absorb fluid and salt like a sponge. There is no virtue in running the fluid in slowly. Meanwhile, blood will be withdrawn for cross-typing and also for serum electrolyte estimations. The haematocrit estimation should also be done. A Foley-type catheter should be placed in the bladder and the hourly output of urine should be recorded. Meanwhile the first litre of Ringer's lactate will be followed up by a second, running somewhat more slowly and as soon as the blood is available 500 cc. of blood will be given as a transfusion. The blood pressure and pulse will be recorded every fifteen minutes. The reason for the blood transfusion is to restore the diminished blood volume as fast as possible in order to overcome the peripheral circulatory collapse; 500 cc. of blood may be adequate but a further 500 cc. may well be required. The first bottle of blood should be given quite rapidly, say over one hour. It is vitally important to restore the blood pressure to normotensive levels as soon as possible, so that renal filtration pressure may be restored and adequate urinary flow re-established. The kidney is the essential regulator of the disproportions between the various electrolytes so that even though the correct proportions of electrolytes are not given, so long as the kidney is functioning well, this disproportion will be corrected. The only way to restore peripheral circulatory collapse is to restore blood volume, and the quickest way of doing this is to give whole blood. If the patient is responding to the therapy, the urinary output will rise progressively hour by hour, the blood pressure will rise, the sensorium will clear, the peripheral cyanosis will go and muscle tone will return. Certainly by the sixth hour the patient should be perfectly fit to go to the operating theatre. By this time he will probably have had 3,000 cc. of Ringer's lactate, 1,000 cc. of blood. This would give him 4,000 cc. of fluid and 530 mEq of Na and 400 mEq of Cl which, as has been seen from Fig. XV, is adequate to repair this initial deficit.

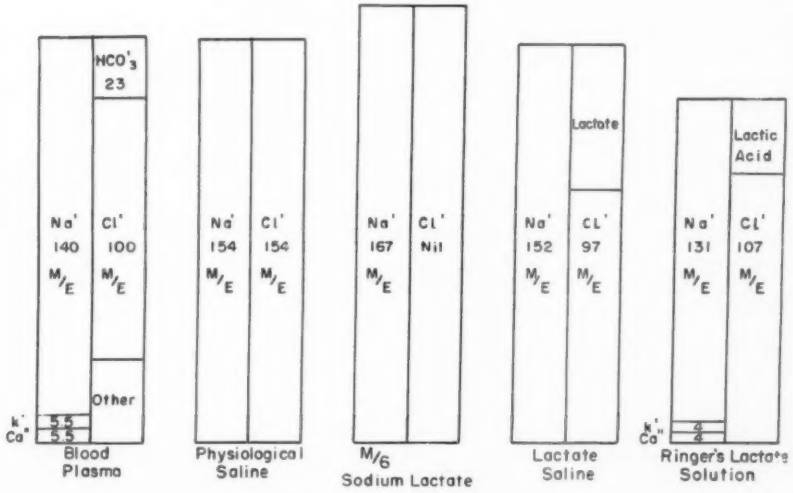


FIG. XVI. Composition of repair solutions.

POTASSIUM METABOLISM

The second part of this paper deals with the present knowledge of potassium metabolism and the practical application of this to surgical patients. Because of the amount of this subject-matter, it will be tabulated.

(1) It is necessary to recall that, in the cell, there is a lot of potassium and very little sodium, whilst in the extracellular fluid there is a lot of sodium and very little potassium (Fig. VIII).

(2) It is now agreed that sodium and potassium can move freely across the cell

membrane. This occurs normally in muscle contraction when the potassium ion carrying the positive charge moves across the membrane to depolarize the cell and so initiate contraction. It also occurs when there is a relative deficit of one or the other ion on either side of the cell membrane.

(3) Of the intracellular potassium, part is bound to the protein of the cell, and part is bound to the phosphate ion and is therefore ionic. The protein bound potassium will ordinarily be retained in the cell unless there is cell catabolism, in which case it also will pass out into the extracellular fluid. This potassium will be in proportion to the amount of non-protein nitrogen also passed out of the cell by the breakdown of the protein. Both this excess potassium and the excess non-protein nitrogen will appear in the urine and therefore will bear a strict relationship to each other.

(4) If there is an excess amount of sodium in the cell or a deficit of potassium then the normal enzymatic processes of that cell will be disturbed.

(5) It is important to realize that the potassium ion is readily excreted by the kidneys — it has a low renal threshold compared with sodium. Therefore as soon as any excess appears in the extracellular fluid it is immediately strained off by the kidneys and as long as renal function is normal, no build-up of potassium in the body can occur. As a corollary, it is perfectly safe to give large quantities of potassium intravenously as long as the renal function is adequate. Indeed potassium citrate has been used as a diuretic for many years. It should also be noted that potassium is readily absorbed from the gut and this is the ideal way to give it in cases of deficiency, as long as oral feeding is possible. Fruit juices and milk will provide large quantities of potassium.

(6) If there is an excessive loss of sodium from the extracellular fluid, then potassium will move out of the cell into the extracellular fluid in order to try and readjust the osmotic relationships across the cell membrane. This occurs therefore in acidosis, but the potassium in the extracellular fluid will be strained off by the kidneys, so that a vast amount of potassium may be lost from the body in acidosis. Furthermore, the levels of serum potassium are no indication of the total potassium deficit of the body. Therefore, it follows from this that in order to repair acidosis due to sodium deficit, such as occurs in diarrhoea or intestinal obstruction, it is vital to give not only sodium but also potassium. This potassium may be given as K Cl and it is usual to add 40 mEq of potassium in the form of K Cl to each litre of normal saline or Ringer's lactate that is given, up to 120 mEq of potassium per day. It is not wise to give more potassium than 40 mEq per litre of intravenous fluid. By this regime a sodium deficit will be much more quickly corrected, because the added potassium will move into the cell and restore the deficit there, thus permitting the Na in the cell to move out into the extracellular fluid and so help to restore the deficit of sodium there.

(7) In alkalosis there is a relative excess of sodium in the extracellular fluid as compared with Cl. This will occur from the vomiting of pyloric stenosis. Under these circumstances some of the excess sodium will move into the cell and displace some of the potassium which moves out into the extracellular fluid and once again will be strained off by the kidneys. Therefore, just as with acidosis, so with alkalosis there is some potassium deficit and in these cases also, potassium should be given to restore the balance along with normal saline. Of course, with the vomiting of pyloric stenosis, potassium is also lost in the vomitus from the stomach, as gastric secretion contains small amounts of potassium.

(8) Because potassium can cross the cell membrane and because it is readily excreted, by good functioning kidneys, it will be evident that the serum levels of potassium are not an accurate indication of the total potassium levels of the body. Thus the potassium levels of the blood may be normal, but there

may be a total potassium deficit. Generally it may be stated that if the serum levels of potassium are low, then there will be a total body potassium deficit, but the reverse situation does not necessarily hold.

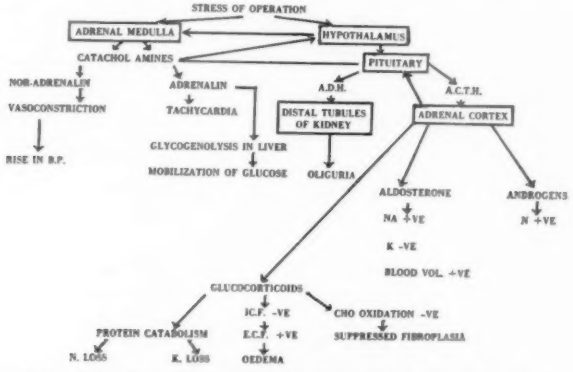


FIG. XVII. The diffuse effects of the stress of operation on fluid, electrolytes and metabolism.

(9) It is evident that for normal muscle contraction and muscle tone, there must be normal levels of potassium and acetylchlorine at nerve-muscle cell junctions. Potassium deficit, therefore, would upset this balance and will result in flaccid muscles which, in the case of the gastro-intestinal tract, will result in loss of tone and paralytic ileus. It is important to realize, therefore, that potassium deficiency may well be a potent factor in post-operative paralytic ileus. Therefore, potassium replacement in the form of K Cl, given in doses of 40 mEq per day should be regularly carried out in the post-operative period, provided there is an adequate urinary output.

(10) The mineral corticoids, in particular aldosterone, have the effect of causing sodium retention and potassium loss. Therefore, in the post-operative period there is an increased loss of potassium, and this should be allowed for by adequate intravenous replacement, if oral intake is not possible. The ideal method of replacing potassium is in the form of milk and fruit juices, but if oral feeding is withheld, then K Cl given as 40 mEq to a litre of fluid, should be used intravenously.

(11) Potassium excess in the extracellular fluid becomes a serious problem in such conditions as severe burns and crush injuries associated with oliguria or anuria. Under these circumstances, due to extensive catabolism, a tremendous amount of intracellular potassium is passed out into the extracellular fluid where it accumulates because the kidneys cannot strain it off. These patients in essence die from potassium excess.

(12) The signs of hyperpotassaemia are:

(i) Lethargy
(ii) Lassitude
(iii) Mental confusion
(iv) Paralysis of muscles of respiration
(v) Paraesthesia of fingers
(vi) Rise of blood pressure at first, followed by a fall
(vii) Brachycardia and heart block.

(13) Signs of hypopotassaemia are:

(i) Generalized muscle weakness
(ii) Flabbiness and hypotonicity of muscles
(iii) Decreased reflexes
(iv) Paralytic ileus and distension of abdomen
(v) Dyspnoea with gasping type respiration.

THE NORMAL EFFECTS OF OPERATION

It is important to appreciate that a surgical operation is a form of stress. Therefore, the normal effects of stress on the fluid and electrolyte balance of the body will take place after any operation. The more major the operation, the more major the stress and the more will the fluid and electrolyte balance of the body be disturbed. Therefore, the post-operative orders for fluids and electrolytes must take into consideration the effects of stress, which have been discussed in the preceding portion of this lecture.

Thus, by referring to Fig. XVII, it will be seen that an operation will increase the amount of anti-diuretic hormone. This will be effective for twenty-four to forty-eight hours, so that after operation there will be a natural oliguria. Therefore, the fluid requirements for the first twenty-four hours should allow for this fact. Hence, for an adult person 1,000 cc. should be allowed for invisible fluid loss and 1,000 cc. for urine output, so that 2,000 cc. should be adequate fluid intake for the first twenty-four hours, provided there is not undue loss by gastric aspiration or from sweating. For the second twenty-hour hours this may be increased to 2,500 cc.

It is wise to keep a patient a little "dry" rather than a little "wet" in the first forty-eight hours after surgery. Furthermore, there should be no undue concern if the patient only passes 300-500 cc. of urine in the first twenty-four hours. There is no indication to catheterize the patient because he has not micturated in the first twelve hours after operation, unless he is uncomfortable and has a distended bladder. The chances are, his bladder will have little urine in it on account of the natural oliguria.

In the first twenty-four hours after operation 5 per cent. glucose in distilled water only should be ordered, and no electrolytes are required unless there has been a previous deficit or unless there is continuing loss from gastric aspiration or fistulae. By referring back to Fig. XIV, it will be seen that operation will cause sodium retention. Therefore, there is no need to give Na Cl in the first twenty-four hours. Potassium should not be given till the oliguria has passed off. Therefore, neither sodium or potassium should be given in the first twenty-four hours. In the second twenty-four hours the basal needs of sodium can be given, i.e. approximately 80 mEq of sodium and, if the urinary output is adequate, 40 mEq of potassium may be added. It need hardly be stated that oral feeding should be resumed as soon as possible because the gut is the best selector of the needs of the body, from the point of view of fluid and salts. After many operations no intravenous fluid or electrolyte therapy is needed and oral feeding can begin forthwith.

SUMMARY

1. The distribution of fluid and electrolytes in the three body compartments has been stated.
2. The regulation of fluid and electrolyte intake and output has been discussed, and the way this regulation may be upset has been pointed out.
3. The calculation of the various fluid and electrolyte deficits is described. The value of the Scribner method of calculating the twenty-four hour loss is stressed.
4. The various repair solutions and their appropriate applications are described.
5. The importance of early restoration of normal renal function is stressed.
6. Potassium metabolism is discussed in a tabulated form.
7. The effects of operation *per se* on fluid and electrolyte regulation are discussed.

REFERENCES

No detailed list of references is given but the following writings and authors have been freely drawn upon:

BLAND, J. H. (1953), "The Clinical Use of Fluid and Electrolyte." Philadelphia, W. B. Saunders.

GAMBLE, J. E. (1947), "Chemical Anatomy, Physiology and Pathology of Extracellular Fluid." Cambridge, Mass., Harvard University Press.

LE QUESNE, L. P. (1954), "Fluid Balance in Surgical Practice." Chicago, Year Book Publishers.

MARRIOTT, H. L. (1947), *Brit. Med. J.*, vol. i, pages 245, 285, 328.

—— (1950), "Water and Salt Depletion." American Lecture Series Publ. 32. Springfield, III, Charles C. Thomas.

MOORE, F. D. (1958), *J. Canad. med. Ass.*, vol. 78, page 85.

MOYER, C. A. (1952), "Fluid Balance." Chicago, Year Book Publishers.

VERNEY, F. B. (1958), *Surg. Gynec. Obstet.*, vol. 106, page 441.

MICRODISSECTION OF SPECIMEN OF HYDRONEPHROSIS WITH COMPARATIVE NEPHRON MEASUREMENTS

By Dora Bialestock

Department of Surgical Research, Royal Children's Hospital, Melbourne

Introduction

DIVERSITIES of nephron structure have recently been emphasized by Bradley and Wheeler (1958). A point has been made by these authors on lack of accurate data for dimensions of human nephrons. They have stated "values in the literature are too few to permit a thorough statistical evaluation". For this reason any figures which might add to the limited knowledge in this field would be of value.

Oliver's work (1939) on microdissection of kidneys from cases of chronic nephritis has shown the diversity of nephron structure and size in cases of long-standing acquired renal disease. He has shown that the kidney units can show enormous variation up to tenfold volume increase of the proximal tubule; atrophic and hypertrophic units occur side by side in the same organ, and that the large well developed glomerular tubules found in these kidneys are supplied by blood which has not passed through a glomerulus. These tubules may be responsible for a good deal of the function in kidneys from chronic nephritis.

Bialestock (1956, 1958) has published descriptions of diversities of nephron anatomy in kidneys of new-born babies. These include nephrons of abnormal convolution with localized dilatations, aglomerular and tubular cysts and abnormal "giant" nephrons.

The present paper aims to describe firstly the observations in an infant aged one month with renal abnormalities; secondly to compare quantitatively nephron dimensions obtained from the case P.D. with figures obtained from the kidneys of two other full-time babes in this age group. One of the kidneys was a normal specimen (J.C.) and the other (R.B.) was the subject of pyelonephritis with hydronephrosis of unknown aetiology.

Materials and Methods

The kidney to be described and the normal specimen were obtained after death at routine post-mortem studies. The specimen from R.B., with hydronephrosis, was obtained at operation (Bialestock, 1958). This specimen revealed its abnormal structure only by micro-dissection. Routine histology gave no indication of the poorly convoluted nephrons, the absence of loops of Henle, and the large bizarre isolated cystic structures seen with ease by microdissection.

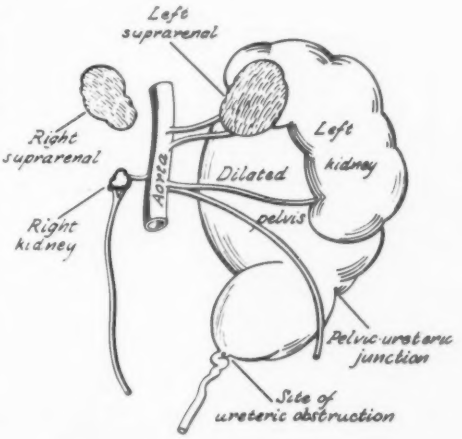
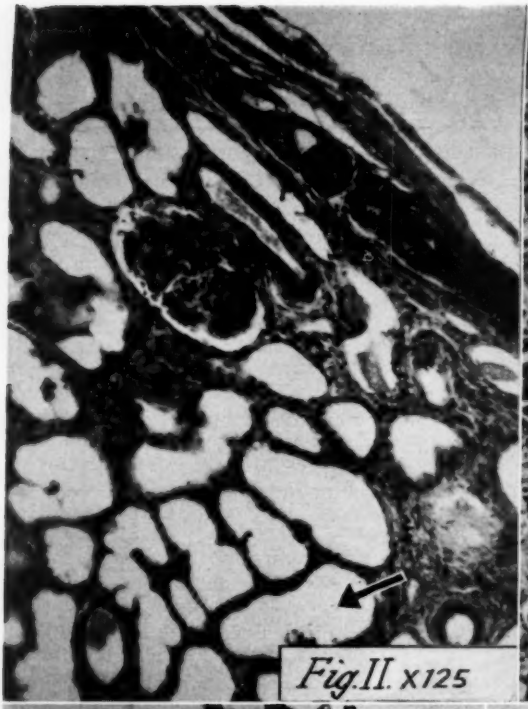
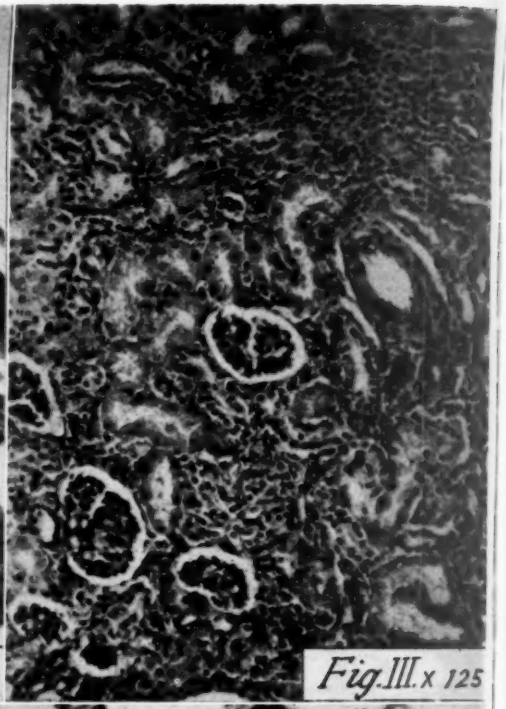


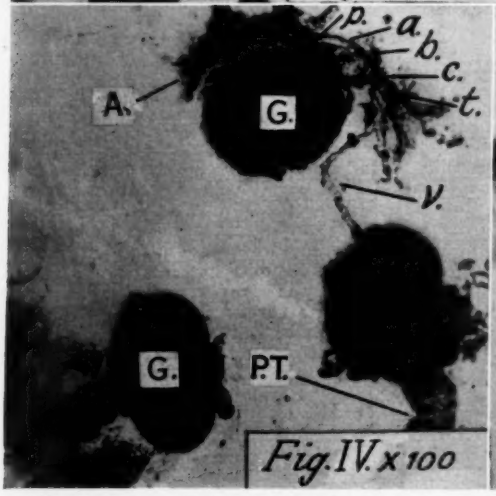
FIG. I. The right kidney represented as a very small yellowish tuft measuring 1.5 cm. x 1 cm. x 0.5 cm. suspended on a very small renal artery. Suprarenal glands were both normally developed.

Material was macerated in concentrated hydrochloric acid following formalin fixation. Details of the method have been published in previous papers (Bialestock, 1953, 1956).

Measurements of the nephrons were made by means of an eyepiece graticule calibrated against a standard milimetre scale.







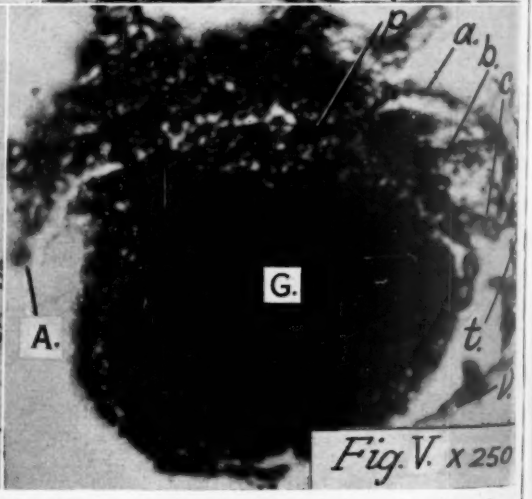


FIG. II. The section from P.D. has been taken in the subcapsular zone to show the plaque of abnormal nephrogenic tissue. The tubular dilatation and hypertrophy seen in this zone was more marked deeper in the cortex. The infiltration of the glomerulus with small round cells and the atrophic remains of a tuft within and oval capsular space (arrow) can be seen at the lower right hand corner.
The degree of hypertrophy of all elements in P.D., more particularly the tubules, can be appreciated when compared with Fig. III.

FIG. III. A section through a normal kidney from a one month old babe. Note (i) the presence of cuboidal epithelium still around the tuft; (ii) the close packing of the glomeruli and tubules with no connective tissue between.

FIGS. IV and V. These pictures illustrate the afferent arteriole (A) at the upper glomerulus (G). This vessel runs along the upper curve of the capsule and enters it at point (p). The efferent arterioles, arising from (p), has three branches (a, b and c) which form a common trunk (t). One branch of this trunk (v) courses down towards the proximal tubule (P.T.) at the right-hand corner. The upper convex surface of the afferent arteriole can be seen to have three distinct branches. These supplied overlying tubules without first passing through the glomerulus.

Case notes of P.D.

History: P.D., a male, was the second child of normal parents with one other healthy child aged three years. The pregnancy was normal, but labour was induced nine days after the due date.

Birth weight was six pounds seven ounces. The mother noted that P.D.'s abdomen was large soon after birth, and that the babe progressed slowly on breast feeds.

Several weeks prior to hospital admission the babe had been miserable, difficult with feedings, crying spasmodically with drawing up of legs, and passing up to ten diarrhoeal motions daily. Five days prior to admission he had commenced vomiting, with refusal of all feeding. Micturition had been normal until four days before admission when no urine was passed; then urine was passed only twice daily since. Twenty-four hours before admission, the child had some difficulty with breathing and several cyanotic attacks.

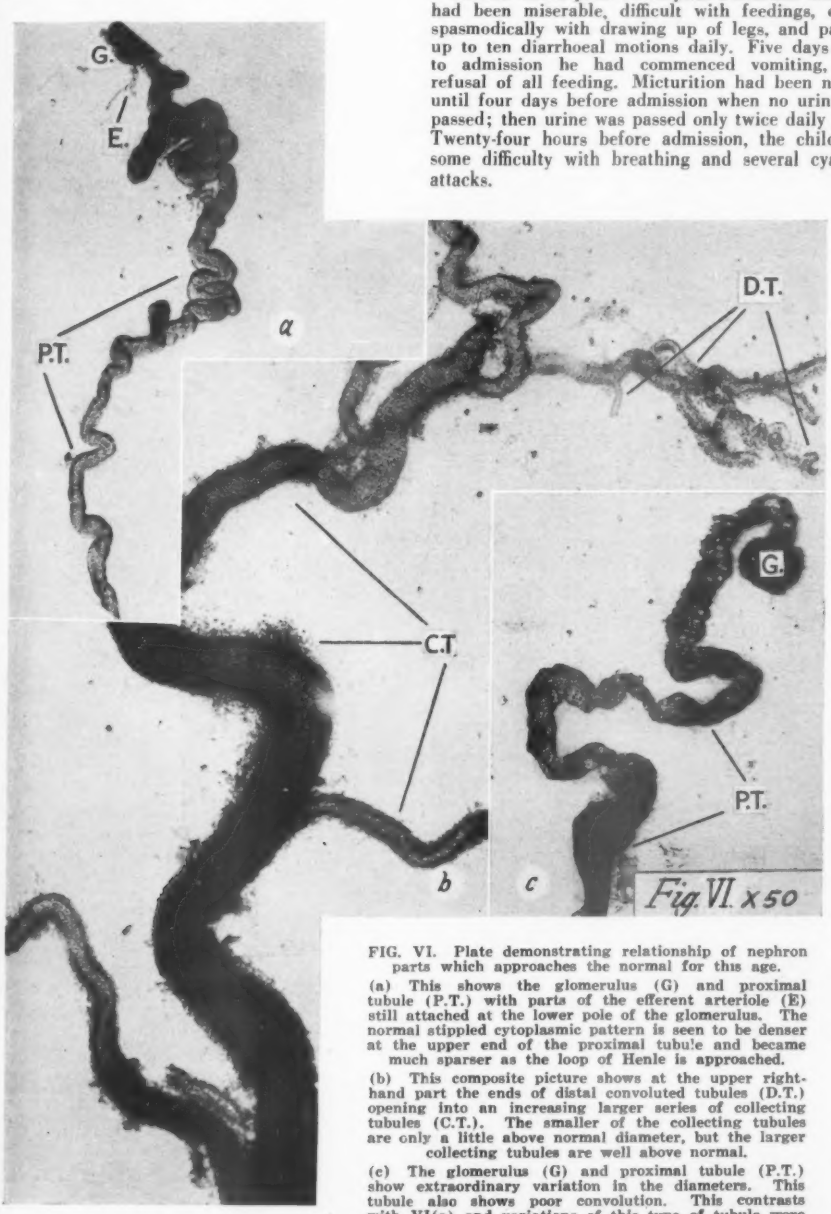


FIG. VI. Plate demonstrating relationship of nephron parts which approaches the normal for this age.

(a) This shows the glomerulus (G) and proximal tubule (P.T.) with parts of the efferent arteriole (E) still attached at the lower pole of the glomerulus. The normal stippled cytoplasmic pattern is seen to be denser at the upper end of the proximal tubule and became much sparser as the loop of Henle is approached.

(b) This composite picture shows at the upper right-hand part the ends of distal convoluted tubules (D.T.) opening into an increasing larger series of collecting tubules (C.T.). The smaller of the collecting tubules are only a little above normal diameter, but the larger collecting tubules are well above normal.

(c) The glomerulus (G) and proximal tubule (P.T.) show extraordinary variation in the diameters. This tubule also shows poor convolution. This contrasts with VI(a) and variations of this type of tubule were found more frequently than the one illustrated in VI(a).

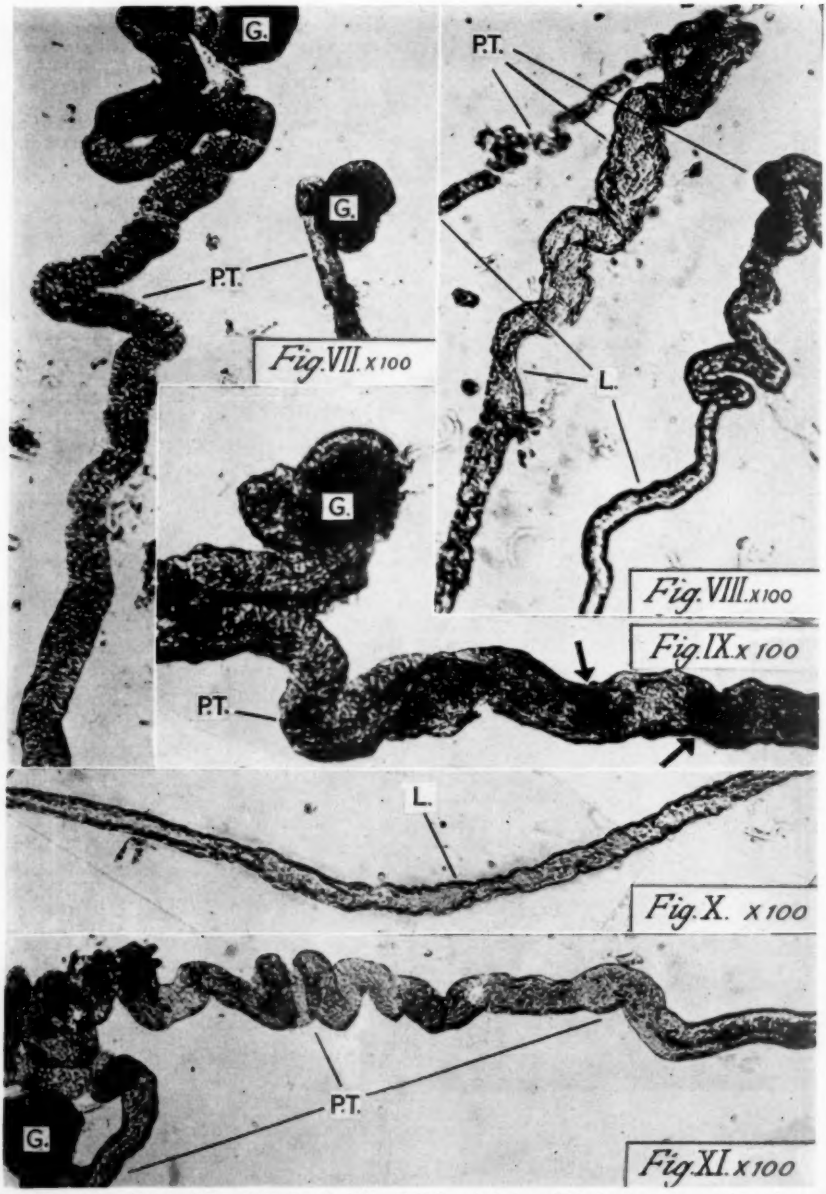


FIG. VII. Glomerulus (G) is followed by the densely stippled cytoplasm which is the common type in proximal tubules. The glomerulus (G) at the right-hand side of this picture is followed by a straight neck segment. The great variation in first parts of the proximal tubule can be appreciated when Figs. VII, IX and XI are compared.

FIG. VIII. The terminations of three proximal convoluted tubules (P.T.) at the beginnings of the straight segment of loop of Henle (L). These extreme variations of dimensions were a common finding.

FIG. IX. The oval glomerulus (G) is followed by a very densely stippled proximal tubule (P.T.). The tubule is widely dilated, in parts equal in diameter to the glomerulus, and only poorly convoluted. Two casts (indicated by arrows) are present within the lumen, but because of the dense stippling cannot be seen as easily as in distal and collecting tubules.

FIG. X. This demonstrates the first part of Henle's loop. This shows calibre and cytoplasmic pattern typical throughout this case.

FIG. XI. The glomerulus (G) is followed by a proximal tubule (P.T.). This picture demonstrates the open type of stippling that can be seen in the cytoplasm of proximal tubules. This stippling is usually found in Henle's loop.

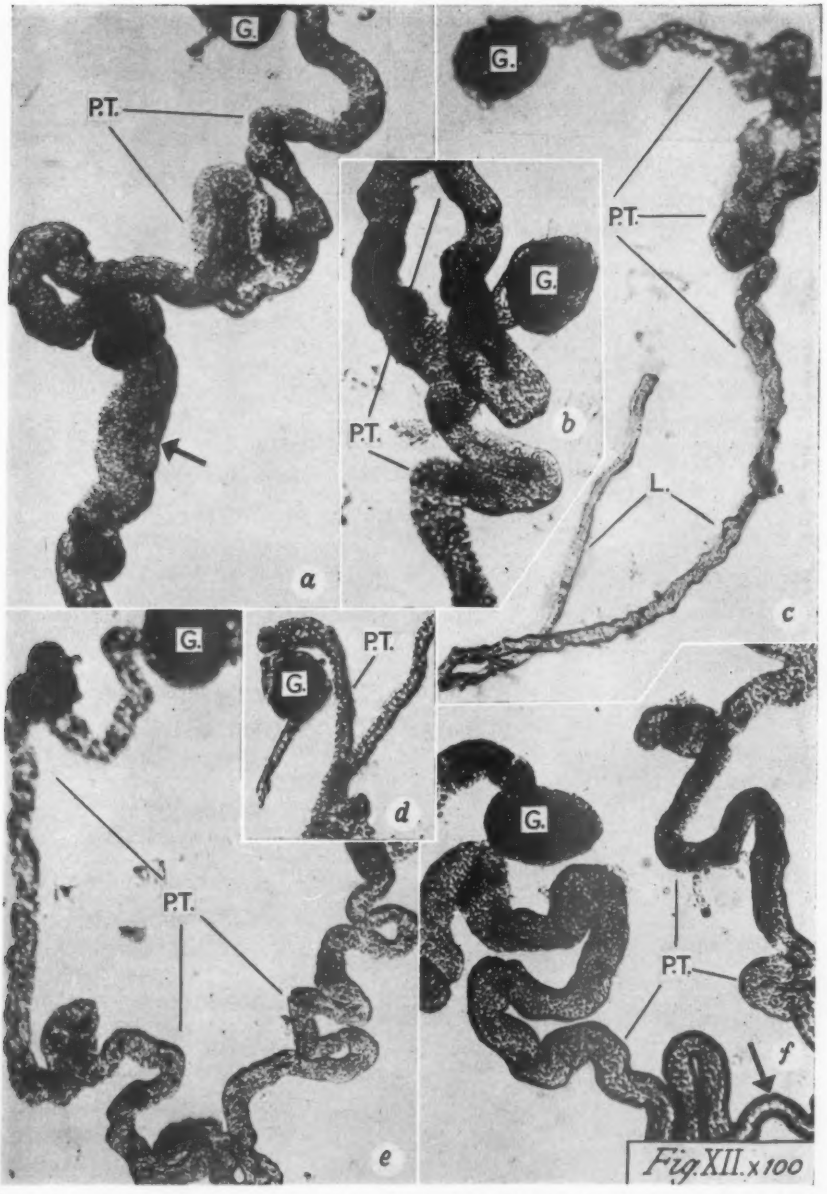


FIG. XII. To demonstrate diameter variations along the proximal tubule and variations in relationship between glomerular and tubular diameters.

(a) The glomerulus (G) is followed by an average proximal tubule (P.T.) until the arrow where sudden unpredictable dilatation can be seen.

(b) The glomerulus (G) is not much bigger than the dilated proximal tubule (P.T.).

(c) The glomerulus (G) is followed by the proximal tubule (P.T.) and loop of Henle (L). The diameter of the glomerulus is about three times that of the tubule and this is the normal relationship. The loop of Henle is different from the adult in that the first part is the wide limb and then the segment narrows.

(d) The glomerulus (G) is smaller than the average and followed by an almost straight narrow proximal tubule (P.T.).

(e) Large glomerulus (G) followed by well convoluted proximal tubule (P.T.) whose diameter is only about one-fifth of that of the glomerulus.

(f) The opposite state of affairs to Fig. XII(a) is demonstrated in this picture, when glomerulus (G) is followed by a proximal tubule (P.T.) which suddenly narrows unpredictably at the arrow.

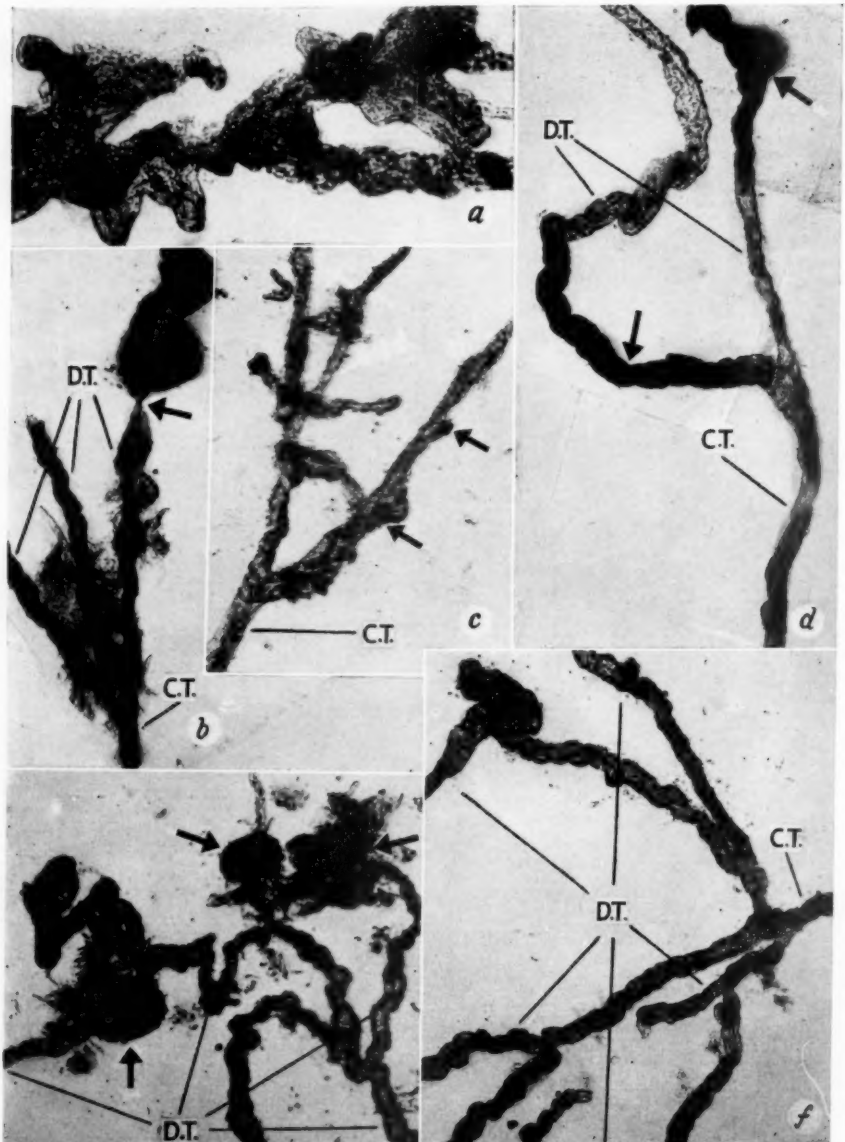


FIG. XIII. Showing changes in distal and collecting tubules.

(a) Small segment of a large collecting tubule. The multiple irregular out-pouchings and diverticulations can be easily appreciated.

(b) The distal tubules (D.T.) opening into a collecting tubule (C.T.). Note the huge dilatation of upper part of the distal tubule, then the constriction (arrow).

(c) Small collecting tubules showing the irregular outline and casts with lumens (arrows).

(d) Distal convoluted tubules (D.T.), and collecting tubules (C.T.) showing the irregularity of diameter and the small diameter of the collecting tubule. Casts are well seen as the dark areas obliterating the normal stippled cytoplasmic pattern (arrows).

(Continued on page 217)

On examination, the only abnormal finding was a large rounded mass occupying the left side of the abdomen.

In hospital the child deteriorated rapidly; his temperature was subnormal (95° F.): he passed only small volumes of urine and began to convulse. The blood urea rose prior to death from 40 mgm/100 cc. to 173 mgm/100 cc. in the six days in hospital.

Post-mortem findings: Relevant post-mortem finding were confined to the genito-urinary tract.

The large left kidney measured 8.4 cm. x 4.5 cm. x 4 cm. The renal tissue was seen as a narrowed rim around the enormously dilated pelvis. There was a stricture in the upper ureter below the pelvi-ureteric junction. The ureter below the constriction was normal in calibre (Fig. I). The blood vessels, coursed around the dilated pelvis, but were not related to the site of constriction at the pelvi-ureteric junction.

Microscopic examination

Microscopic section of the large left kidney revealed considerable diminution in number of nephrons when compared with a section from the normal infant of the same age (Figs. II and III). Gross dilatation and hypertrophy of glomeruli and tubules was also a feature. There was an increase in intertubular connective tissue. Infiltration with small round cells was evident throughout the renal substance, involving glomerular tufts in some instances, and obliterating the capsular space. Other glomeruli showed only atrophic remnants of the capillary tufts and marked cystic dilatation of the capsular space. The arterioles and the small collecting tubules were within normal limits.

Scattered as discreet plaques beneath the capsule of the kidney were the remains of abnormal nephrogenic tissue (Fig. II).

To summarize:—On routine microscopic examination one could say that the kidney was definitely abnormal, with diminution of the total number of nephrons. Glomeruli and tubules seen were larger than normal, but small collecting tubules and blood vessels were within normal limits. The nephrogenic tissue persisting as discreet plaques was abnormal.

The usual finding if nephrogenic tissue is still present, is a layer right around the kidney just beneath the capsule; not, as in this case, a number of discreet plaques. According to Potter (1952) this zone should have disappeared by the time the foetus weighed 5.5 lbs.

Microdissection findings

To appreciate the changes along the length of the nephron and the relationships of abnormal structures, the kidney was subjected to micro-dissection and measurement. As soon as micro-dissection was commenced the extraordinary diversity of nephron changes far greater than previously observed was evident.

Atrophic changes, more difficult to see, and even more difficult to demonstrate photographically, were often obscured by grossly hypertropic dilated tubules. Changes, as succeeding photographs well illustrate, were sporadically scattered, bizarre and unpredictable. Average nephrons dissected in this specimen of P.D. (Figs. VIa, VIb, VII, IX, XI) were enlarged well beyond the normal for this age. Glomeruli varied in diameter from 96μ to 256μ with gradations between these two figures. In about half the glomeruli observed and measured, the shape was oval; in the others it was round. Tufts were seen to vary from a dark mass almost filling Bowman's capsule to a small condensation of material in an otherwise dilated cystic space.

The neck, or part of the proximal tubule immediately below the glomerulus, was seen to vary considerably in structure, from a localized narrow thin portion, the "swan neck" of Clay *et alii* (1953) to a ballooned section, or to the flabby wide tubule not distinguishable from the rest of the proximal convolution. The "swan neck" has not been observed to be a feature distinctive for any one condition in my material. Proximal tubules were of the flabby large dilated type, but localized zones of constriction or dilatation not related to the presence of debris within the lumen were seen frequently.

Whereas in the normal cases the tubular diameter was one half to one third that of the glomerulus, in this case tubular diameters were often as wide as those of the glomeruli.

(e) A group of distal tubules (D.T.) with the round dark abscesses (arrows) intimately attached. The tubules below these inflammatory masses are normal.

(f) Distal tubules (D.T.) joining to form a collecting tubule (C.T.). The irregular outline with small diverticulation is seen in these tubules. The diameter varies considerably along the length of the tubules. The diameter of the first order of collecting tubules (C.T.) was as small as or smaller than those of the dilated hypertrophied distal tubules (D.T.).

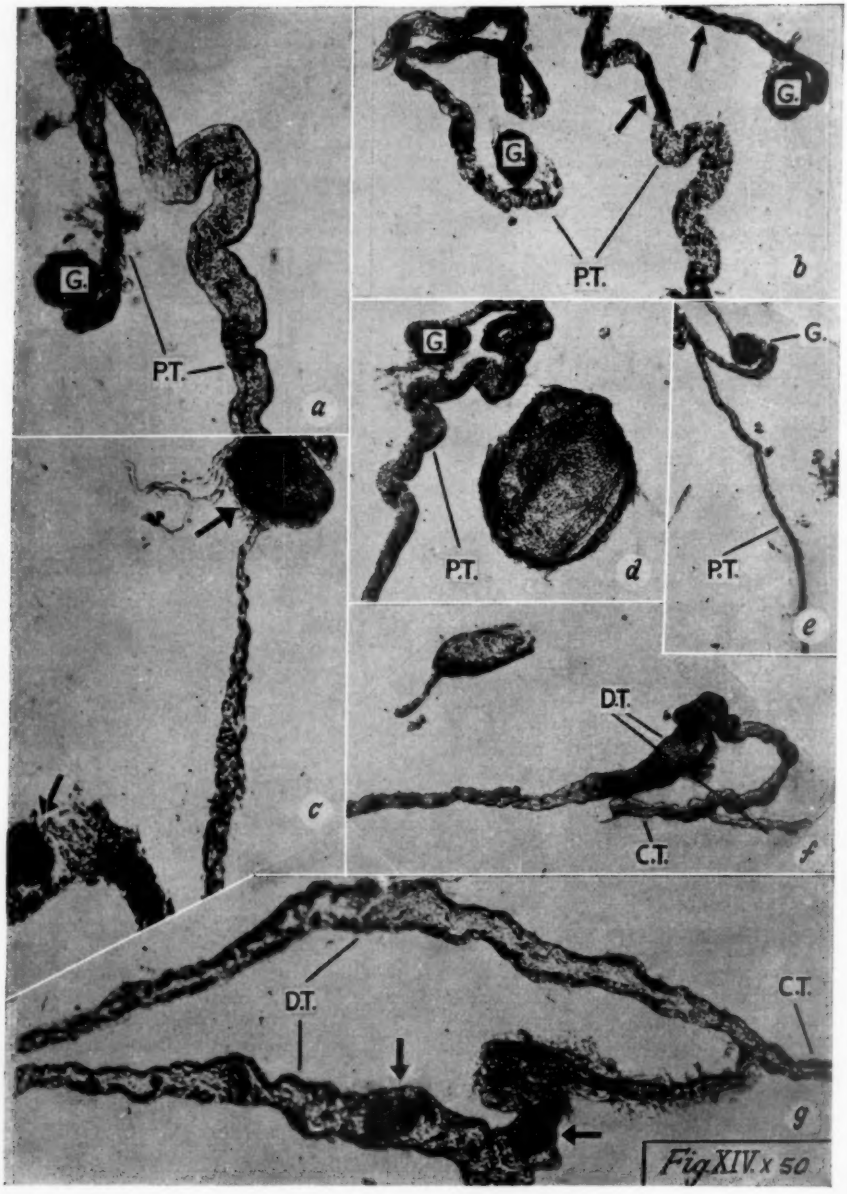


FIG. XIV. Demonstrates gross abnormalities.

(a) The glomerulus (G) is followed by a long narrow segment comprising the first part of the proximal tubule (P.T.). The rest of the proximal tubule is flabby, poorly convoluted and the diameter is about the same as that of the glomerulus.

(b) Upper ends of two nephrons — left-hand one with average configuration, right-hand tubule shows a very long narrow segment adjacent to the glomerulus, followed by a normal segment, then another narrow segment (arrow) followed by a large widely dilated part.

(c) Two grossly abnormal structures found in the medullary part of the cortex. They were imbedded in dense inflammatory material. The cysts (arrows), seen at the upper ends of the straight tubules of varying diameter, were possibly of glomerular origin. These bizarre units opened directly into the collecting tubules. (Continued on page 219)

A few nephrons with the normal relationship were seen (Fig. XIIc). Loops of Henle, in contrast with R.B. (Bialestock, 1958) were in this case well developed, both long and short loops being found in about equal proportions. Structure of the loops were more uniform in diameter and showed fewer localized abnormal changes than seen in other parts of the nephron.

Distal convoluted tubules showed considerable variations, especially common being localized segments of irregular out-pouching and generalized hypertrophy.

The diagram (Fig. XVII) and the measurements demonstrate the overall increase in length as well as diameter seen in most nephrons above that found in the normal and near normal cases.

The smaller collecting tubules, though they featured the localized irregularities of contour showed very little dilatation or hypertrophy. Large collecting tubules were tremendously hypertrophic (Fig. XVII).

Localized masses of precipitated material within lumens of the tubules were very commonly seen. In proximal tubules there is denser protoplasmic stippling and these blur the outlines of casts within. In distal and collecting tubules, the casts could be defined sharply, as outlines were not obscured by the dense protoplasmic pattern. In no instance was a cast observed to produce dilatation above nor collapse below. This would suggest that complete occlusion by these intratubular deposits must be a rarity. Cysts and abnormally convoluted nephrons, in contrast to those in R.B., were in this case often smaller than the hypertropic nephrons, though some of the glomerular cysts were large (Figs. XIVd, XVa and c).

The inflammatory areas, seen as dark discrete patches adherent to tubules, more particularly the distal and collecting tubules, could be, in most instances, dissected from the tubules leaving the underlying parenchyma unharmed. Less commonly the fine granulations involved the tubular basement membranes and could not be removed without destroying the tubular continuity (Figs. XVIa, b, c, d).

Of great theoretical interest is the demonstration illustrated in Figs. IV and V of the large extra-glomerular arterioles which can be seen even at the age of one month in this abnormal kidney. In a recent article (Bialestock, 1957), the case for the extra-glomerular arterial supply to renal tubules was reviewed and the occurrence of such blood vessels in a normal adult without chronic renal disease was demonstrated.

Measurements of the nephrons obtained by microdissection

In the following measurements the figures for the normal J.C. and for R.B. were, in the case of glomeruli and proximal tubules, obtained by averaging about fifty measurements. Average figures were justified as the range of variation found was not very great. Dimensions for other parts of the nephrons were more difficult to obtain accurately and the figures given here are only the averages of, at the most, five measurements in each case. Dimensions of P.D.'s nephrons are recorded seriatim as this will best demonstrate the extraordinary variations found.

Convoluted tubules had to be unwound to considerable degree for measurement of length and the stretch put on the tubules could be a source of error. However, as the same person carried out all measurements, this would probably be a constant factor and would not affect the comparative values.

The diagram (Figs. XVIIa and b) was constructed from the average measurements of Table 1.

(d) A glomerulus and proximal tubules, average in configuration for this case (note oval glomerulus), alongside a large round isolated cyst probably of glomerular origin.

(e) At this lower magnification the nephron which is the same as that of Fig. XII(d), can be seen in full extent. The almost complete absence of convolution and the regularity of diameter makes it very different from the average in this case.

(f) The uppermost structure is a cyst of glomerular origin attached to a small narrow tubular remnant which ends blindly. The lower part of the picture demonstrates the widely dilated distal tubule (D.T.) at the left-hand side and the more normal distal tubule (D.T.) at the right-hand side combining to form a collecting tubule (C.T.). The localized cystic dilatation, and the isolated cyst are morphologically similar.

(g) Two distal convoluted tubules (D.T.) demonstrating the localized dilatations and tortuosities of contour found in these tubules. Well defined casts can be seen (arrows). The diameter of the collecting tubule (C.T.) into which they both open at the right-hand side of the photograph is smaller than parts of the distal tubule.

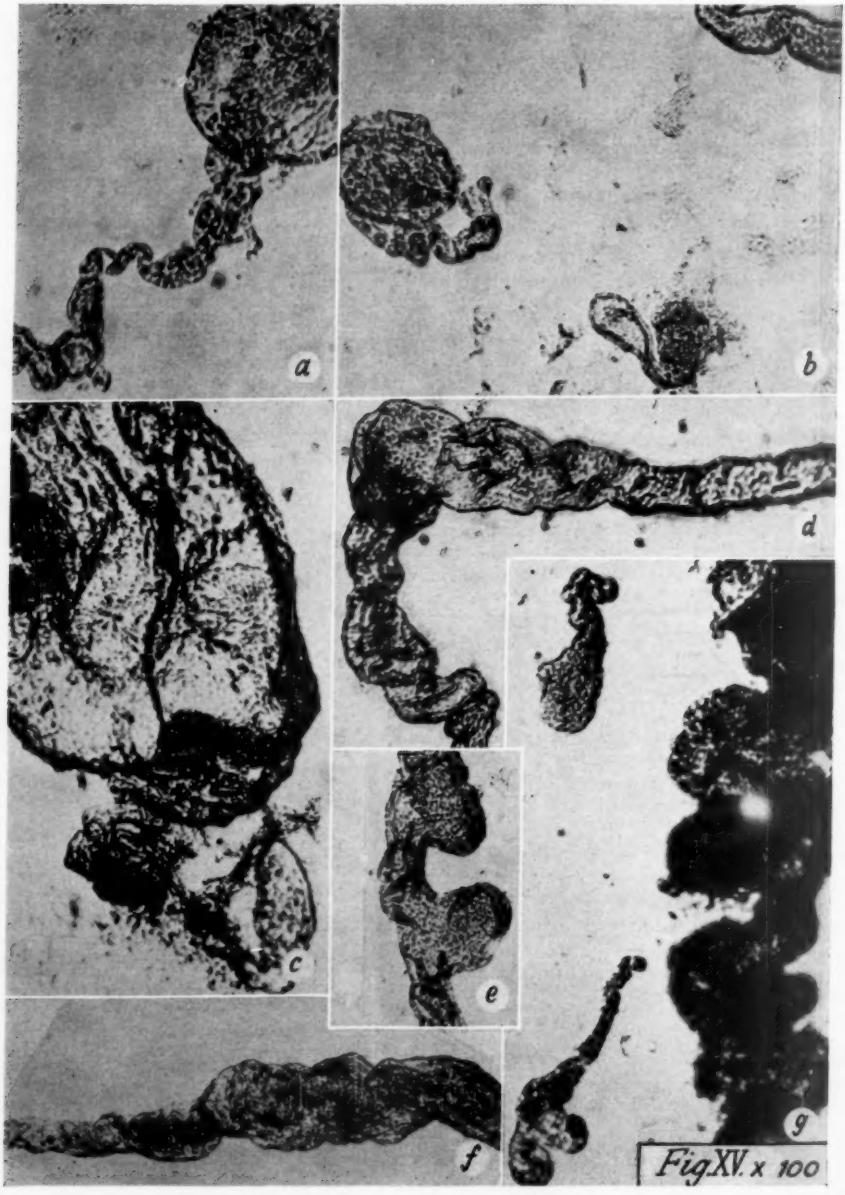


FIG. XV. Localized cystic dilatations and discrete cysts.

(a) An abnormal structure which is probably the remains of a nephron. The right-hand side possibly represents the remains of Bowman's capsule. The blood vessels are attached to the outside of this structure which was embedded in granulation tissue. The narrow irregular tubule ends as a clubbed blind sac.

(b) Small abnormal structures with possible origin as nephrons, ending as blind tubules. The relationship to these and the average tubule can be seen by the tubule included in the upper right-hand corner.

(c) A large round cyst attached to a short narrow tubule with widened clubbed end.

(d) and (e) These views of unpredictable localized dilatations along distal tubules with sudden narrowing are possible transition stages to isolated tubular cyst formation.

(f) Diverticulation of distal tubule.

(g) These small irregular blind ending structures cannot as easily be related to nephrons. Note the hypertrophic tubule along the right side of the photograph. An idea of comparative sizes can be appreciated.

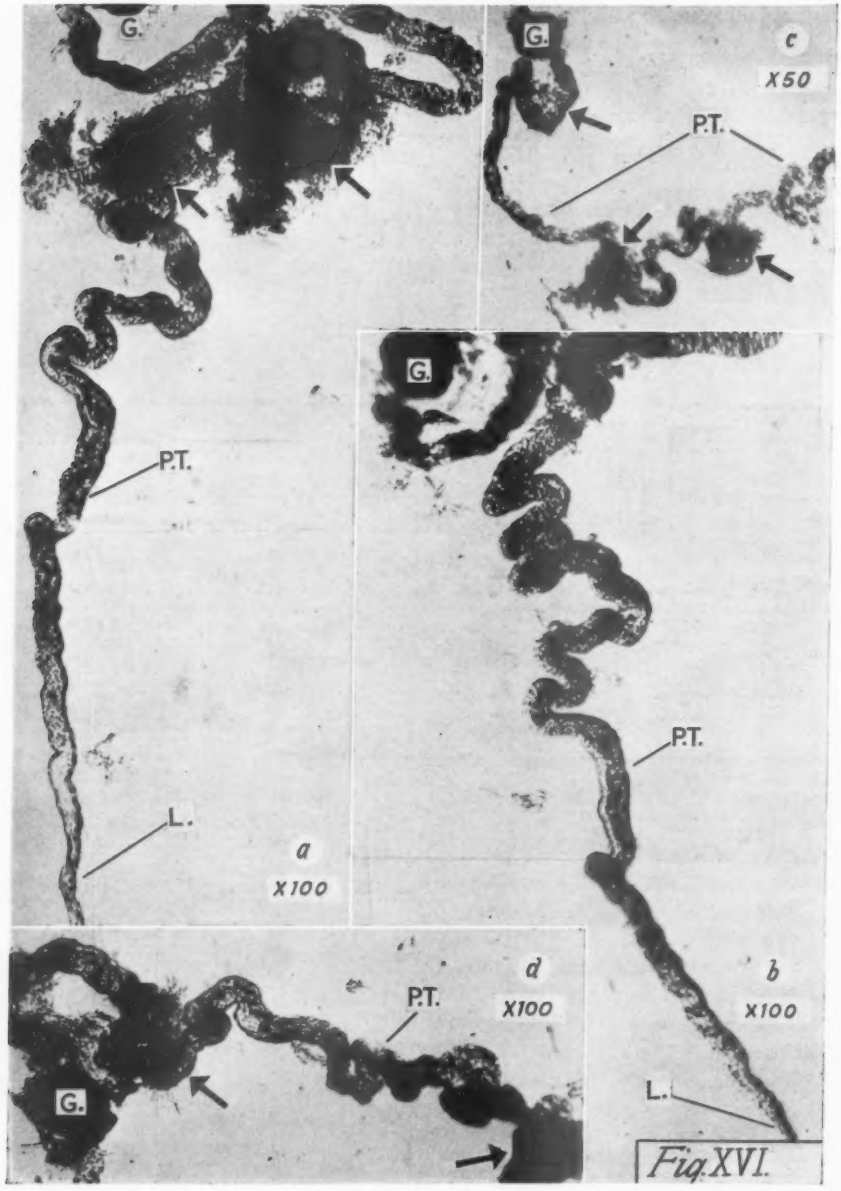


FIG. XVI. Demonstrates relationship between inflammatory tissue and the nephron.

(a) and (b) These two pictures are of the same nephron (the glomerulus in Fig. XVI(a) is nearly out of the picture but its lower third is well seen). These demonstrate the granulation tissue involvement of the loops of the proximal tubule, seen as the black amorphous tissue overlying the tubule in Fig. XVI(a) (arrow). The next picture shows how the nephron has not been involved in that the tubule continuity is unharmed. Casts are present in this tubule lumen but cannot be outlined as easily as in the distal and collecting tubules.

(c) and (d) In contrast to the above illustrations, the granulation tissues (arrow) in these two nephrons could not be removed without disrupting the nephron entirely.

Measurements of J.C. — Normal.

Glomeruli subcapsular 75μ Deep 90μ.

Proximal tubules (shorter subcapsular 20μ-30μ x .9 mm.-1.2 mm.

(deeper) 30μ-45μ x 1.8 mm.-2.4 mm.

Loop of Henle thick limb 15μ x .6 mm. thin limb 6μ x 1.8 mm. Distal tubule 30μ x 1.2 mm.

Collecting tubule 30μ-45μ x 1.5 mm., 60μ x 1.2 mm. 105μ x .3 mm.

(5) 130μ x 195μ x 2.73 mm. x 39μ-104μ.

(6) 130μ x 130μ x 3.25 mm. (incomplete) x 65μ.

(7) 104μ x 130μ x 3.64 mm. (incomplete) x 65μ.

(8) 118μ x 117μ x 1.95 mm. (complete) x 39μ.

(9) 130μ x 130μ x 3.9 mm. (halfway along thin segment) 54μ-13μ.

(10) 156μ x 156μ x 3.9 mm. (no thin segment) x 65μ.

TABLE 1

AVERAGE FIGURES

	Normal 1 month J.C.	*R.B. (mildly abnormal)*	*P.D. (grossly abnormal)*
Glomerulus	82μ	75μ	169μ
Proximal Tubule	30μ x 2 mm.	25μ x 2.4 mm.	83μ x 3.42 mm.
Loop Thick	15μ x .6 mm.	25μ x .8 mm.	46μ x 1.6 mm.
Loop Thin	6μ x 1.8 mm.	none present	22μ x 2.38 mm.
Distal Tubule	30μ x 1.2 mm.	none measured	53μ x 4.78 mm.
Collecting Tubules	37.5μ x 1.5 mm. 60μ x 1.2 mm. 105μ x .3 mm.	75μ x .9 mm. 90μ x 1.3 mm. 150μ x .36 mm.	43μ x .256 mm. 68μ x 1.56 mm. 455μ x .91 mm.

R.B., one month old babe with hydronephrosis and pyelonephritis

Glomerulus subcapsular 60μ, deep 90μ.

Proximal tubule 20μ-30μ x 1.8 mm.-.3 mm.

Loop of Henle, only rudimentary thick segments were seen.

Only two measured 20μ x .9 mm., 30μ x .72 mm.

Distal tubules—no measurements recorded.

Collecting tubules 75μ x .9 mm. 90μ x 1.3 mm. 150μ x .36 mm.

Cystic structures 195μ x 180μ, 180μ x 240μ, 300μ x 720μ, 300μ x 300μ attached to straight tubule 60μ x .72 mm.

P.D. One month

Glom. *P.T.*

(1) 208μ x 156μ x 2.47 mm. x 65μ-156μ.

(2) 130μ x 169μ x 1.3 mm. x 65μ-117μ.

(3) x 5.2 mm. x 39μ-117μ.

(4) 130μ x 156μ x 3.34 mm. x 65μ.

(11) 156μ x 156μ x 3.25 mm. x 54μ.

(12) 130μ x 130μ x 4.55 mm. x 65μ.

(13) 192μ x 128μ (cyst) x 3.2 mm. x 32μ.

(14) 160μ x 128μ x 4.438 mm. 86μ-24μ (loop average diameter 64μ).

(15) 128μ x 154μ x 3.20 mm. x 64μ.

(16) 192μ x 192μ x .64 mm. x 12μ (very abnormal).

(17) 256μ x 256μ x .768 mm. x 12μ-16μ (very abnormal).

(18) 154μ x 154μ x 4.68 mm. x 64μ-43μ.

(19) Small glomerulus 96μ x 96μ.

Segments of loops of Henle

(thin) 13μ x .780 mm. (about half of loop only)
13μ x 40 x 3.2 mm. (entire loop) long

(thick) 65μ x 1.92 mm. (entire loop) short

(thick) 26μ x 1.28 mm. (not complete)

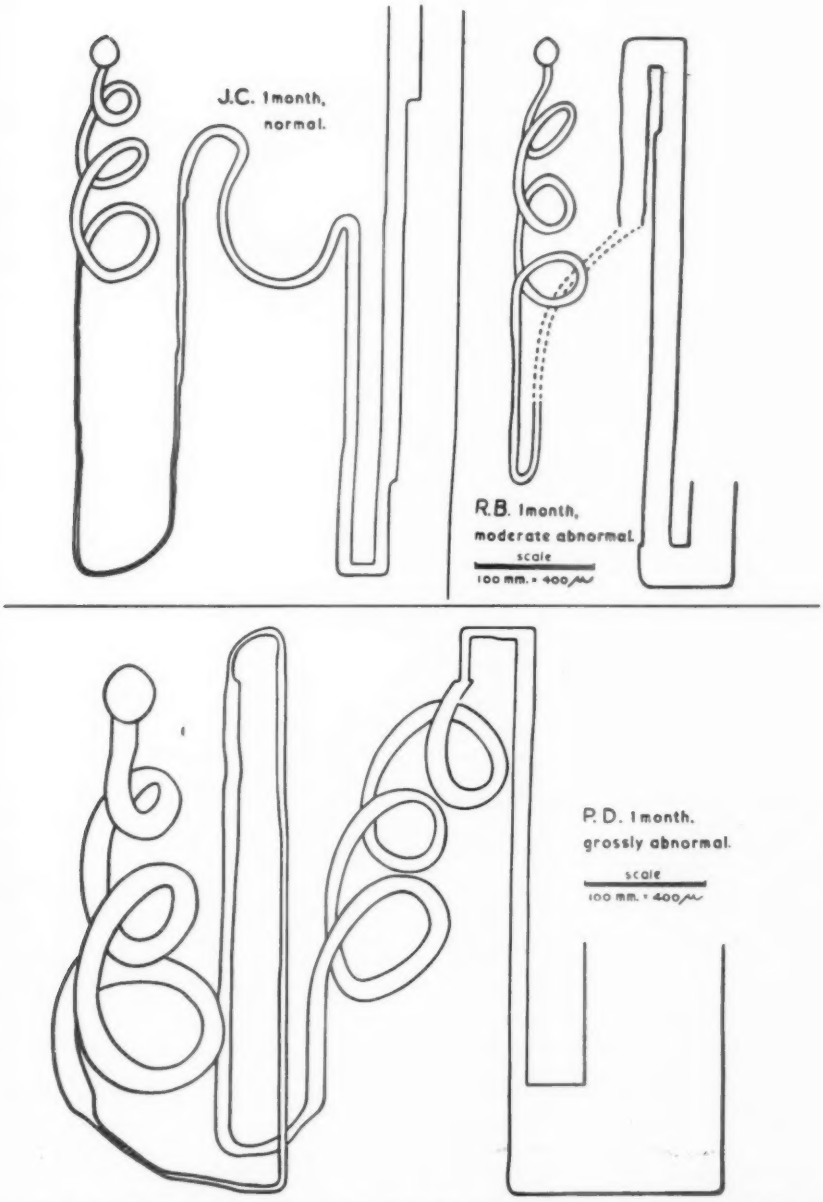


FIG. XVII. The diagrams of the nephrons of J.C., R.B. and P.D. have been drawn to scale from average figures for nephrons given in Table 1.

Relative lengths and diameters can be compared and glomerular, tubular relationships appreciated. In the normal case J.C. note the presence of all parts of the nephron and the relationship of diameters with gradually increasing diameters of the collecting tubules. In R.B., there were no loops of Henle present. The distal tubules were not accurately measured and not included in this diagram (dotted segment). In P.D. note the overall increase in length and diameter of all components. The narrow first order of the collecting tubules contrasts with the huge dilation of the largest collecting tubule.

Distal Tubules

26μ x 6.4 mm.
26μ x 64μ x 5.12 mm.
26μ-64μ x 2.816 mm.
} complete

26μ-104μ x 2.73 mm. — not quite complete.

26μ-130μ — length not complete.

Collecting Tubules

43μ x .256 mm., 64μ-72μ x 1.08 mm., 43μ-64μ x 2.048 mm., 455μ x .910 mm.

Abnormal Structures

Cyst 104μ x 286μ; Cyst 78μ x 234μ; Cyst 192μ x 288μ.

DISCUSSION

From the detailed study of P.D. it is apparent that a baby with grossly abnormal genito-urinary system can develop normally to full term and can thrive reasonably in its early weeks. Urinary tract infection, which must have been occurring insidiously throughout the life of this babe, was present until just before death, without symptoms or signs.

From the observations made on P.D., what clues have emerged to suggest the mode of formation of these bizarre nephrons? Can they be explained by a simple mechanical theory or is there a common factor which caused widespread damage to many parts of the urinary system at one stage of development? Does compensatory hypertrophy account for the changes in the left kidney and what effects on the renal parenchyma stem from the obstructive element of the ureteric stricture? Finally, has inflammation been a cause of these diverse anomalies of structure within the kidney?

The widespread distribution of the abnormalities including rudimentary right kidney, enlargement of the left kidney with intra and extra-renal hydronephrosis, and constriction of the ureter, could not be explained by any simple mechanical theory.

The presence of both the abnormal nephrogenic zone and the bizarre unpredictable hypertrophic and hypotrophic changes, on the other hand, points to a common factor acting at a time in the development of the kidney in utero.

The factors responsible for the multiplicity of renal abnormalities must have been general, because the entire genito-urinary system has been involved, but also local, because one kidney was rudimentary whilst the other was hyperplastic, but with diminution of number of nephrons.

Compensatory hypertrophy no doubt played some part in the enlargement of the structures in this infant (P.D.), but the measurements disclose diameters and lengths far beyond those recorded by other workers for simple compensatory hypertrophy.

Compensatory hypertrophy has been shown to occur in experimental animals and in humans (Echart, 1888; Mineru Arataki, 1926; Oliver, 1924; Addis, 1950) when a kidney or part thereof has been removed. Where figures were available these indicated that increase in size was of an order to compensate for tissue removed.

It seems, therefore, that the variations in size of the nephrons cannot be accounted for by compensation alone.

Furthermore, the extraordinary diversity of hypoplastic and hyperplastic nephrons seen in this case were not found by the other workers in the compensatory hypertrophy experiments.

The factor of osbtruction must now be taken into account. This may have been responsible for the atrophic changes and perhaps for the overall diminution in the number of nephrons.

The inflammatory changes, though widespread, severely disrupted continuity of tubules in only the minority of instances. These changes did not seem in any way related to the formation of the bizarre abnormal nephrons.

Animal experiments have been performed showing that external factors can cause congenital renal anomalies and that these anomalies can be reproduced in the subsequent generations. Bagg (1925-1926) exposed mice to small doses of unscreened X-ray. Bagg's experiments included 1,800 post-mortems and 285 showed absence of a single kidney. This was the commonest visceral abnormality produced.

Bagg found in adult mice the unilateral kidney was hypertrophic, but in the newborn the single kidney was not hypertrophic. From this he concluded that the hypertrophy occurred in the post-natal period. One hundred and forty-nine of the animals showed complete agenesis and these were all born alive. Others had congenital hydronephrosis and polycystic kidney.

Thomas *et alii* (1957), by feeding of the insecticide diphenylamine to rats, produced cystic kidneys.

That renal abnormalities can be produced by external stimuli and transmitted to future generations, is of paramount significance when an attempt is made to look for aetiological factors. The importance of long family histories, perhaps extending over many generations, might be of value in assessing the causation of anomalies in human material.

The occurrence in this infant of the cystic changes, derived from glomeruli or tubules or both, again supports the thesis that cyst formation is derived from a general factor acting on the kidney during development as enunciated in a previous communication (Bialestock, 1956).

The presence of well-developed extraglomerular blood vessels to tubules in a babe of only one month of age would point to the autonomy of the tubule which should be capable of functioning even if the glomerulus is absent or destroyed (Bialestock, 1957).

Comparative measurements in this paper are few in number, but when a great many more are accumulated, it is hoped that as Hamilton (1958), has pointed out, precision will increase with the increasing number of repetitions.

SUMMARY

1. In a baby, P.D., aged one month who died in renal failure, microdissection of the nephrons in the single large abnormal hydronephrotic kidney, associated with definite organic ureteral stricture in the upper ureter, revealed an extraordinary variation in structure from hypoplastic nephrons or parts thereof to the immensely hyperplastic units. These structures and their measurements are compared with those of a normal kidney and a kidney showing moderate hydronephrosis of unknown origin.
2. Greatest enlargement was in length and diameter of the convoluted tubules and in the diameter of the largest collecting tubules.
3. A gradation between almost normal elements to the grossly hypertropic nephrons, which made up the majority of the nephron population, was seen.
4. Localized abnormalities, scattered sporadically in a bizarre fashion along the length of a tubule were frequently seen.
5. Not only were there nephrons of grossly abnormal size, but also nephrons with gross abnormalities of structure. Gradations between these abnormal nephrons and isolated cystic bodies of glomerular, but also of possible tubular origin, were common.
6. Extensive infection in the peritubular connective tissues, without necessarily involving the continuity of tubules passing through such granulation tissues, was the usual finding.
7. Cast material was frequently seen within tubular lumens, more clearly in collecting tubules. These casts did not appear in any instance to block tubules completely. No collapse below nor dilatation above the site of the material was seen.
8. Quantitative nephron dimensions from three babes aged one month illustrate the enormous variation that can occur even at this young age.
9. The existence of extraglomerular blood vessels to tubules in this abnormal kidney was demonstrated.

ACKNOWLEDGEMENTS

I would like to thank Mr. E. Thake of the Visual Aids Department of the University of Melbourne for the drawings of Figures I and XVII and for his aid in assembling the photographs.

KEY TO ILLUSTRATIONS

1. All photographs of kidney structures are enlargements from 35 mm. negatives. The magnification scale on the prints refer to these. The prints

throughout are enlargements to one and a half times (approximate) from the magnifications indicated.

2. Photographs have been used to demonstrate anatomical detail and comparative sizes only.

G — glomerulus
P.T. — proximal convoluted tubule
L — loop of Henle
D.T. — distal convoluted tubule
C.T. — collecting tubule
A — afferent arteriole
E — efferent arteriole
a, b, c — branches of efferent arteriole
t — trunk of efferent arteriole
v — branch of efferent arteriole

REFERENCES

ADDIS, T. (1950), "Glomerulonephritis", The Macmillan Company, New York.

ARATAKI, M. (1926), *Amer. J. Anat.*, vol. 36, page 437.

BAGG, H. J. (1925-26), *Amer. J. Anat.*, vol. 36, page 275.

BIALESTOCK, D. (1956), *J. Path. Bact.*, vol. 71, page 51.

—— (1957), *Anat. Rec.*, vol. 129, page 53.

—— (1958), *Aust. Ann. Med.*, vol. 7, No. 2, page 93.

BRADLEY, S. E., and WHEELER, H. O. (1958), *Amer. J. Med.*, vol. 24, page 692.

CLAY, R. D., DARMODY, E. M., and HAWKINS, M. (1953), *J. Path. Bact.*, vol. 65, page 551.

ECKARDT, C. Th. (1888), *Virchows Arch. Path. Anat.*, vol. 114, page 245.

HAMILTON, M. (1958), *Lancet*, No. 7028, vol. 1, page 977.

OLIVER, J. (1924), *Arch. Int. Med.*, vol. 34, page 258.

—— (1939), "Architecture of the Kidney in Chronic Bright's Disease", New York, Paul B. Hoeber Inc.

POTTER, E. L. (1952), "Pathology of the Fetus and the Newborn", Chicago, The Year Book Publisher Inc.

THOMAS, J. O., and COX, A. J. (1957), *Stanford Med. Bull.*, vol. 15, page 90.

PHYSIOLOGICAL PRINCIPLES IN THE USE OF THE CARDIOPULMONARY BYPASS*

By Henry T. Bahnson and Frank C. Spencer†

Baltimore, Maryland, U.S.A.

THE successful use of an artificial heart-lung machine to allow cardiopulmonary bypass and intracardiac surgery has increased surgical interest in physiology, particularly that of the blood, the blood gases, and their transport in the cardiovascular system. The principles involved in the pump or artificial heart and the oxygenator or artificial lung, cover a wide spectrum of physiology. Much of this physiology is well known and simply needs application to this specific problem, but new problems have arisen regarding trauma to blood, circulation and oxygen consumption under anaesthesia, the pulmonary circulation, and post-operative convalescence.

We propose to discuss these physiological principles in connection with design of apparatus and with conduct of the bypass as we have seen them in 185 clinical perfusions, eight of which were done during a visit to the Royal Prince Alfred Hospital, Sydney, N.S.W.

The Pump

A number of somewhat similar pumps are in general use. Gibbon (1954), who began work on an artificial heart-lung machine in 1932, adopted the DeBakey roller pump, and this or a modification of it is widely used and has many advantages. Dependent upon the action of a roller to force blood along a tube this simple device eliminates the use of internal valves, is associated with little trauma to the blood, and is capable of a wide range of output depending upon the size of the tubing and the speed of the roller.

A simple device and one which is quite popular, probably because it is used in industry and is readily available commercially, is the Sigmamotor finger pump used throughout the wide experience of Lillehei and his associates (1957). This depends upon a milking action of the multiple fingers; it can be accurately controlled. As in the roller pump, flow is to some extent determined by filling pressure as diastolic filling is dependent on elastic recoil of the tubing and spring tension on a backing plate. It is slightly more traumatic in causing hemolysis than the other pumps mentioned, but at the present has probably been used in a greater total number of perfusions than any other type of pump throughout the world.

A more pulsatile pump with a regulated input and output is significantly gentler on the blood, particularly if some means of external valving is introduced. The presence of internal valves, although tolerated, imposes additional problems of disturbance of the flow stream, clotting of the valve, and cleaning. Several fine accurately controlled pulsatile pumps have been developed, those used by Dennis (1956), Melrose (1959) and by Crafoord and Senning (1957) being exemplary.

A worthy aim in development of equipment for cardiopulmonary bypass has been to duplicate the functions of the body in so far as possible. Maintenance of a pulsatile flow was one of the first such functions to be abandoned in the interest of simplicity and expediency. There is scant experimental evidence to indicate that the pulse is not necessary in the maintenance of the body economy, as measured by renal function or by survival of the animal and maintenance of blood volume (Weslowski *et alii*, 1955). Scores of perfusions with minimally or nonpulsatile pumps attest to the fact that adequate perfusion can be obtained without a pulse. The crucial clinical or animal experiments have not yet been reported which might support the conjecture that an adequate perfusion could be improved or that flow in the finer ramifications of the arterial system would be

---

*From the Department of Surgery, Johns Hopkins University and Hospital, Baltimore, Maryland.

†Aided by Grant H-1326, National Heart Institute, U.S. Public Health Service.

better if a pulse were introduced. Thus if we assume that the best mechanism has been developed over the eons of our time, we must assume on abandoning the pulse in our ideal machine that the pulse present in the mammalian circulation is necessary to allow the myocardium to rest (not a problem with electrical power) or that possibly the alternating pulsatile flow is more easily controlled than a continuous one.

All equipment used in handling blood must inflict upon the blood as little trauma as possible. The commonly used measurement is that of hemolysis. Performance with a tolerable amount of hemolysis is readily obtained and in fact the body will tolerate large amounts of hemolysis if the free hemoglobin and the more dangerous ghosts of the red cells are presented to it gradually. Because of this it is impossible to estimate the true hemolysis of perfusion equipment if an animal is in the circuit as it effectively filters the products of red cell destruction. Flink (1947) has shown that in the normal dog levels of free hemoglobin as high as 3,700 mg. per cent. may be tolerated without renal damage. Although tolerated, hemolysis is obviously undesirable and lack of trauma in the handling of blood is one of the first requisites in pump construction. Projecting edges, rapid acceleration or deceleration, restricted orifices, and high or low pressure shock waves are some of the more obvious causes of hemolysis.

Given a mechanically sound pump decision must be made in regard to removing and returning the blood. In the body the heart is in direct contact with large venous conduits, the cavae, from which it fills without significant loss of pressure. With cardiopulmonary bypass two considerations require that some resistance be imposed between the cavae and the mechanical heart. First the pump must be removed from the operative field and connection be made by tubing; second, the multiple connections require that cannulae be placed before the bypass is started and hence non-obstructing or only partially obstructing cannulae must be used so that flow can continue past them until the complete cardiopulmonary bypass is begun. The latter requires that small diameter tubing and the former that a length of tubing be introduced both resulting in a loss of pressure head. Hence, some means of applying suction on the venous side must be used. If, while on the bypass, less than a pre-bypass resting flow is to be pumped, one may obstruct the venae cavae about the cannulae and be assured of an adequate reservoir behind the obstruction and a constant flow. With this method the venous pressure must necessarily rise. If, on the other hand, one aims to pump a flow approaching that of the resting level some means must be employed to prevent aspiration of the caval walls into the cannula and fluttering of flow when the suction and flow are not accurately balanced. The method which we and most others employ is that of gravity drainage. In essence, as a siphon this substitutes a constant pressure removal for the constant volume removal of the directly coupled venous pump. Since pressure head is dissipated with the frictional resistance and this loss increases as the flow increases, progressively less suction is applied at the caval end as the flow is increased. Even with gravity flow if the frictional loss in tubing is not great, as in large diameter tubing, and the siphon head is greater than needed, the cavae will be drawn into the cannula intermittently and chattering will occur. This is not as damaging to the cava, however as if a constant volume pump is used as in the latter an inordinately high pressure may be developed and the cava damaged. The difference would seem to be one of degree, the natural force of gravity being in this case the gentler.

Arterial return similarly introduces problems of pressure and flow. The normal heart pumps into a large diameter aorta but our accepted requirement that a bypass be set up in parallel necessitates the use of a relatively small cannula inserted into one of the arterial branches. The larger the cannula the less the resistance to flow and the less trauma will be inflicted from turbulence through the small diameter tube. The subclavian and femoral artery are most commonly used, and selection of one or the other is largely a matter of personal preference. Our preference at present is for the femoral; it is easy of access and of good size. In either instance blood flows retrograde in the valveless aorta to its base, thus nourishing the heart through the coronary arteries and of course supplying flow throughout the body.

We shall return to discuss the volume of blood to be pumped and its control in a later section, but the desired pump must have an output from 300 to 5,000 millilitres per minute, possibly with a change of fittings for the high or low range in the interest of economy of the blood required to fill the apparatus.

THE OXYGENATOR

There is at present no evidence that the oxygenator must serve any function other than that of respiration, the addition of oxygen and the removal of carbon dioxide. The other functions of the lung such as that of a filter, a blood reservoir, and a producer of antibodies can all be temporarily dispensed with. The normal adult human at rest takes up about 135 millilitres of oxygen per square metre of body area, or about 250 millilitres for an average size man, and disposes of about 80 per cent. of this volume of carbon dioxide. Under anaesthesia and the conditions of intracardiac surgery metabolism is reduced and requirements are lowered by about one-fourth. The subject will survive with considerably less than this volume, just as he will survive brief haemorrhagic shock or starvation of other essential nutrients, but with a normal and adequate flow the oxygen consumption when on cardiopulmonary bypass should be similar to that of the resting conditions just prior to bypass. The normal adult human lung has a surface area of about 100 square metres, roughly the size of a tennis court, which is used for exposure of blood to the alveolar air and which is obtained by the multitude of small alveoli and capillary surfaces. Fortunately, in constructing an oxygenator we need much less area than this because of the large reserve in the human lung, the fact that we can ventilate with pure oxygen instead of air (which is 21 per cent. oxygen), and the fact that the patient is at rest and at a slightly sub-basal level of metabolism. Because of its great solubility and rapid diffusibility carbon dioxide presents few problems with any of the oxygenators. Oxygen diffuses less rapidly and this is the reason for attempting to get a large surface area in a small volume. The desired large surface area has been obtained by the use of stationary or revolving screens, by cylinders, or by discs revolving and dipping in a reservoir of blood (Allen *et alii*, 1958). All of these methods involve the display of a thin film of blood over a large surface area. The bubble oxygenator, on the other hand, obtains a large surface area by the introduction of macroscopic bubbles of oxygen into the blood. These are later removed by the use of a silicone antifoam preparation and by passage through a slow-moving reservoir in which the bubbles rise to the surface. Bubble oxygenators are extremely efficient in oxygenation and can be regulated to control simultaneously the carbon dioxide, but their safety depends upon the reliability of removal of bubbles. This is less certain at high flow rates, so that bubble oxygenators have usually been employed at less than resting flow. Our early clinical experience was with the De Wall (1957) bubble oxygenator, but since changing to the stationary screen type of Gibbon we have more safely used higher flows.

There is widespread enthusiasm for a membrane oxygenator with the hope that separation of the blood-gas interface by a plastic membrane may result in less damage to the blood, and that such an oxygenator would allow a closed system of nearly constant volume. In spite of widespread endorsement of the membrane oxygenator as the oxygenator of the future, the advantage of the closed system is partially lost by the necessity of aspirating cardiac venous flow from the open heart; it seems impossible to do this without introducing bubbles. At present the membrane oxygenator, being developed most actively by Clowes (1958), requires a large filling volume and is not sufficiently efficient nor easily handled for widespread use.

Considerable interest recently has been expressed in poisoning by oxygen as a cause of some of the unexplained difficulties with perfusion (Effler *et alii*, 1959). The damaging effect of an atmosphere of pure oxygen on metabolism in vitro has been clearly established, and there is evidence of damage to the lungs of the intact mammal after a number of hours exposure to an atmosphere of oxygen. Harm can be caused in a shorter time with a higher oxygen pressure. A possible means of oxygen poisoning can be visualized if one considers that the tempera-

ture of the bypass apparatus and its contained blood tends to approach room temperature unless vigorous efforts are made to maintain it higher. Oxygen is more soluble in cold than in warm blood. Hence, if oxygenation occurs at a cool temperature and blood is pumped back to a warmer subject, oxygen will tend to come out of the blood and will exert a higher partial pressure. Thus, partial pressures of oxygen in the range known to cause oxygen poisoning may be produced. The role this plays in clinical perfusion has not been clarified but reason indicates steps must be taken to prevent an extraordinarily high oxygen pressure. To control this it is necessary to maintain the temperature of the oxygenator near that of the subject or to regulate the load on the oxygenator so that complete saturation of the oxygenated blood does not occur. Then a slight rise in temperature of the blood on entering the patient will result in saturation of hemoglobin rather than the exertion of a harmfully high oxygen pressure. Some degree of oxygen unsaturation of the blood returned to the patient, though not desirable, seems satisfactorily tolerated. In large patients who tax the limit of our screen oxygenator we have occasionally worked with arterial saturations between 70 and 90 per cent without evident detriment, provided flow to the patient was kept high and near the normal resting cardiac output.

Of importance in the oxygenator but receiving less attention is the removal of carbon dioxide. Carbon dioxide presents few problems because it so readily diffuses and can be removed, but some regulation of this gas must be maintained as it is an important agent in regulation of cerebral vasomotor tone and cerebral circulation. Especially when there is a low arterial pressure lack of carbon dioxide will result in cerebral ischemia and probable damage. Our practice with the screen oxygenator is to ventilate the apparatus with 2.5 per cent carbon dioxide in oxygen. This usually results in a partial pressure a little less than normal. A slightly lower than normal partial pressure of carbon dioxide, however, is tolerated and in fact anaesthesia for a cardiopulmonary bypass involves hyperventilation throughout the procedure with the accompanying low pressure of carbon dioxide.

CONDUCT OF THE BYPASS

Apparatus and its manner of use have improved together but there seems little question that equipment is available with which bypass may be continued safely for longer than an hour. The manner in which this is conducted, however seems almost as important as the apparatus used. A leading question has concerned the use of "high" or "low" flows. The low flow proponents got off to a commanding lead with the development of controlled cross-circulation by Lillehei and his associates.

From the start of his work in 1932, Gibbon had aimed at bypassing with a flow equal to that of the patient before the bypass. The present trend is to a return to this ideal which is now more safely achieved with improved apparatus. It has been demonstrated that rate of survival for perfusions longer than half an hour is reduced and acidosis with accumulation of fixed acids is greater with flows less than about 60 millilitres per kilogram, or approximately 1.6 litres per square metre of surface area (Litwin *et alii*, 1959). This accumulation of fixed acids may not be evident until 4 to 6 hours after operation but appears related to the volume of flow used during the bypass and its duration (Effler *et alii*, 1959).

For these reasons proper flow, we believe, is approximately that which the subject is pumping prior to the use of the bypass. The normal resting cardiac index is reduced from about 3 to 2.5 litres under the effects of anaesthesia and the open chest. This is the range of flow which can be expected and which can be obtained if no obstruction to venous outflow is placed. If venous return is allowed to enter the apparatus unrestricted, the volume flow will be determined largely by the circulating blood volume. Kirby and associates (1958) have nicely demonstrated the need for a larger circulating blood volume with increasing flow; this might be viewed as an increment to distend further the vascular space.

The problem of what parameter should be used to control the flow through such an apparatus remains unsettled; the answer at present is largely dependent upon the type of equipment used. If a low and restricted flow

is used, flow can be set and a constant volume pumped without collapsing the venae cavae. With a higher flow, however, a controlled gentle suction is required or some means of gravity drainage must be used as mentioned above. The latter introduces an open system with a blood-gas interface, and flow control is needed. A frequently used method is the regulation of output from the machine to match venous inflow so that the volume of blood in the apparatus remains constant. This can be done either manually or automatically. Irrespective of the mechanism of control the volume of the blood in the apparatus must be reckoned with so that the patient is neither unknowingly bled nor transfused. Fine moment to moment regulation of the volume in the machine does not seem necessary if an adequate reservoir of blood is used, however, for if the flow is simply set at a desired high level, such as 2.5 litres per square metre surface area, then blood which is pumped to the patient will come back by the venous cannulae unless the latter become obstructed. The practice of setting the arterial pump at a desired flow and allowing any small variation in venous return to be compensated by withdrawing from a reservoir in the bottom of a lung has much to recommend it as thus any temporary pooling of blood in the heart or chest which would cause a decrease in venous return would not cause a reduction in return to the patient.

We routinely monitor arterial and central venous pressure through catheters inserted through the femoral vessels. Of the two, maintenance of a nearly normal venous pressure seems more important.

Intravascular pressures during the bypass have varied considerably and their level has at times been confusing. Even with flows of 3 litres per minute the mean arterial pressure often is a little lower than before the bypass in our experience. Two possible explanations for this are suggested: one, that venous pressure is lowered when siphon gravity drainage is applied to the cavae through large cannulae and this is reflected back through the vascular tree (central venous pressure usually falls a few millimeters of mercury) or, two, that systemic flow before the bypass is started is lower than that mechanically maintained when bypass is started and additional blood is required to fill the system for the higher flow. The latter explanation seems to be the usually proper one in our experience. We attempt to maintain central venous pressure at about the pre-existing level.

Moderately close control of the temperature of the blood has been attempted with the bypass, largely because of the danger of cardiac arrhythmia with hypothermia. Since the needs of the organism are diminished with lower temperatures it is quite possible that the apparatus of the future will have a temperature control as well as a flow control, as suggested by Sealy and associates (1957). Early experience with a combination of hypothermia and perfusion appeared to combine the dangers of both without adding their advantages. With greater familiarity with both modalities the combination of the two has been made safer. Oxygenators capable of supplying an adult at normal temperature are available so that cooling to reduce oxygen consumption of the whole organism is not necessary. The reduction of oxygen consumption with cooling of the heart may be an important consideration, however, as interruption of coronary flow may be necessary for the repair of some cardiac defects, notably aortic valvular lesions. In such cases prolongation of the period the aorta may be safely opened greatly lessens the technical problems associated with coronary artery cannulation.

Cardiac arrest may be induced during bypass in order to obtain a quiet heart, to diminish its metabolism if the aorta is opened and coronary flow stopped, or to diminish the cardiac venous return to make the operative field less obscured. The more physiological means of doing this is with acetylcholine (Lam *et alii*, 1955) or one of its longer acting derivatives, such as mecholyl. Arrest can be obtained by the use of these drugs but the heart usually continues to respond to touching and stimulation, even with large doses. For this reason the use of potassium, as suggested by Melrose (1955) and clinically employed by Effler, is more effective.

Potassium injected into the coronary circulation causes an arrest of the heart in diastole when a level of 1 mg. potassium citrate

per millilitre of blood or about 10 milliequivalents of potassium per litre is obtained in the perfusing fluid (Bentall, 1958). When the level of potassium is reduced, systole begins again. In practice potassium citrate in oxygenated blood is injected into the ascending aorta until arrest occurs. To restart the heart after the repair is completed the aorta is reopened and the potassium-rich blood flushed out. Cardiac action resumes slowly but with increasing vigour. Duration of arrest should be as short as possible, the safe limit often being given between 20 and 40 minutes (Lam *et alii*, 1955). Arrest for periods up to fifty-eight minutes has been used in our experience with survival. This is a procedure which, though tolerated, and perhaps beneficial in minimizing trauma to the heart during repair is not ideal and may have important harmful effects. Helmsworth and associates (1958) demonstrated a high level of potassium (30 mEq. per litre) in blood in the heart during potassium induced arrest and showed that this concentration injected into an isolated appendage caused myocardial necrosis. Ventricular necrosis was also seen in dogs with potassium induced asystole maintained on a bypass (Helmsworth *et alii* 1958) and this was more striking than in animals treated similarly but with arrest induced by ischemia alone. The opponents of induced arrest, and we consider ourselves as such, prefer to tolerate the contraction during repair rather than accept the potential damage of increased potassium or cardiac ischemia. If need be, the aorta can be cross-clamped briefly to allow a more blood-free field during difficult suturing. On the basis of our experience our current practice is to induce arrest, and this by ischemia, only when the aorta must be opened for a short time, as in treatment of children with congenital aortic stenosis. When a longer period is required, as in adults with aortic stenosis, we prefer to perfuse the coronary arteries. We do not now use potassium induced arrest.

When blood is exposed to a foreign surface (and this might include any surface other than that of endothelium) clotting begins. This tendency is greater with unclean surfaces, less with clean non-wettable surfaces such as that offered by some plastic materials or a siliconed surface. Clotting also is accelerated with stagnation even within the body as demonstrated by clotting between a point of arterial occlusion and the nearest entering artery. In equipment used for extracorporeal circulation everything possible is done to diminish chemical trauma by the use of non-wettable plastic materials, high polish of steel surfaces, and avoidance of seams which may collect protein; in some instances coating with a silicon preparation may be used. Flow is kept moving and corners in which blood may stagnate are avoided. Most systems use a filter to trap any bits of fibrin which may have formed, although filters themselves may introduce additional trauma.

Some means of rendering the blood incoagulable is obligatory, and heparin has adequately filled this need. Blood for priming the machine is drawn the morning of operation and is treated with 40 milligrams of heparin per litre. Approximately this same concentration is used in the blood of the patient, the dose of heparin being 3 mg. per kilogram of body weight (normal blood volume is approximately 85 ml. per Kg). After the bypass is completed heparin can be neutralized by addition of protamine. Since neutralization can be accomplished with heparin antagonists there is probably greater risk in using an inadequate amount of heparin than too much since with inadequate heparinization widespread thrombosis and fibrinolysis may occur and fibrinogen be depleted.

POST-OPERATIVE CARE

Post-operative care has required a sharpening of sensitivity to minor complications and tightening of management. The liberal volume of fluids given the adult after gastrectomy would promptly lead to fluid retention and failure in the child who has had his ventricular septal defect repaired. We have followed the lead of others, notably the group at the Mayo Clinic (Sturtz *et alii*, 1957), in sharply restricting fluid for the first day to 500 millilitres per square metre of surface area with half again this much the second day and 1,000 millilitres the third and subsequent days. Before such restriction was given electrolyte disturbances were common, particularly lowering of sodium and chloride.

About a gram of salt is given per square metre beginning on the second or third day. After this, ordinarily, fluids will be taken by mouth and regulation is easier. Greater care in administering fluids is undoubtedly predicated upon lessened reserve in their handling by the heart which has been traumatized and also because of the shifts in fluid spaces associated with correction of the cardiac defect.

One of the most important considerations and one of considerable physiological interest concerns the role played by increased pulmonary vascular resistance displayed by most patients with congenital heart disease and particularly those with ventricular septal defects. Especially in patients with a high resistance prior to operation the convalescence may be precarious. Small catheters have been left in the pulmonary artery and left atrium by our group in order to obtain samples of mixed venous and arterial blood. Oxygen content of these samples has been used with measurement of oxygen consumption to estimate the cardiac output. In patients who are severely ill and in a precarious state the cardiac index frequently is less than half of the lower limits of normal. These studies have been helpful in uncovering a low cardiac index before the usual clinical signs would suggest it and allow one readily to distinguish between pulmonary and circulatory causes of difficulty. A lowered mixed venous oxygen saturation indicates a low cardiac output and suggests the use of digitalis, diuretics, further limitation of fluids, or venesection. On the other hand, slight unsaturation of the arterial blood or that from the left atrium would suggest a respiratory deficiency and suggests the use of high oxygen breathing, tracheotomy to diminish respiratory resistance and allow for more ready removal of secretions, and in some instances the use of a respirator.

Arterial pH has been measured routinely and has been helpful in demonstrating the presence of a respiratory or metabolic acidosis. Respiratory acidosis suggests a mechanical aid to ventilation, such as a respirator and metabolic acidosis has more frequently been seen with a diminished cardiac output.

Another physiological tool is occasionally required in the treatment of patients with atrioventricular heart block. An A-V block may be produced by placement of a suture or trauma to the bundle of His. Although a child with a ventricular rate in the range of 50 to 70 might be able to survive satisfactorily under normal conditions, in the early post-operative period he needs a more rapid rate. Hence, when block is present it is customary to implant an electrode into the myocardium which is used with unipolar stimulation to drive the heart at any desired rate (Brockman *et alii*, 1958). For a child the rate around 100 to 110 seems necessary and for an adult a rate between 80 to 100. With an electrode implanted on the heart and a neutral electrode on the skin shocks of 3 milliseconds with from 0.2 to 10 volts have worked satisfactorily. For the temporary heart block this may be lifesaving and in many instances heart block is only temporary. It has been our experience that it has only prolonged life, however, in patients who do not revert to a normal rhythm as none of the several patients whom we have discharged from the hospital with a heart block, and in whom the block has not reverted within several months, is now alive. The slowed rate is not tolerated by these children although patients with congenital heart block seem to thrive satisfactorily.

CONCLUSION

Intracardiac surgery, made possible by the use of a mechanical heart-lung apparatus, has leaned heavily upon our fund of knowledge of cardiovascular physiology, and, indeed, successful intracardiac operations are not possible without an understanding of this basic physiology. Surgery's debt to physiology will be amply repaid, however, as the temporary substitution for the normal heart of a mechanical one, whose output can be varied at will, allows investigation of animal and human hemodynamics which was not previously possible. Such investigations should benefit both disciplines.

REFERENCES

ALLEN, J. G., MOORE, F. D., MORROW, A. G. and SWAN, H. (1958), "Extracorporeal Circulation." Proceedings of a symposium sponsored in Chicago, 1957, by National Heart Institute. Springfield, Ill, Charles C. Thomas.

BENTALL, H. H. (1958), in Allen *et alii*, "Extracorporeal Circulation."

BROCKMAN, S. K., WEBB, R. C. and BAHNSON, H. T. (1958), *Surgery*, vol. 44, page 910.

CLOWES, G. H. A., Jr. and NEVILLE, W. E. (1958), in Allen *et alii*, "Extracorporeal Circulation."

CRAFOORD, C., NORBERG, B. and SENNING, A. (1957), *Acta Chirurg. (scand.)*, vol. 112, page 220.

DENNIS, C. (1956), *Surg. clin. N. Amer.*, vol. 36, page 423.

DEWALL, R. A., WARDEN, H. E., VARCO, R. L. and LILLEHEI, C. W. (1957), *Surg. Gynec. Obstet.*, vol. 104, page 699.

EFFLER, D. B., KOLFF, W. J. and GROVES, L. K. (1959). *Surgery*, vol. 45, page 149.

FLINK, E. B. (1947), *J. Lab. clin. Med.*, vol. 32, page 223.

GIANELLI, S., ROBINSON, J. H., BEST, R. J. and KIRBY, C. K. (1958), *Surgery*, vol. 44, page 378.

GIBBON. J. H. (1954), *Minn. Med.*, vol. 37, page 171.

HELMSWORTH, J. A., SHABETAI, R. W. and MARGOLIAN, J. (1958), *J. thorac. Surg.*, vol. 36, page 214.

——, KAPLAN, S., CLARKE, L. C., Jr., MCADAMS, A. J., MATTHEWS, E. C. and EDWARDS, F. K. (1959), *Ann. Surg.*, vol. 149, page 200.

LAM, C. R., GEOGHAGEN, I. and LEPORE, A. (1955), *J. thorac. Surg.*, vol. 30, page 620.

LILLEHEI, C. W., WARDEN, H. E., DEWALL, R. A., STANLEY, P. and VARCO, R. L. (1957), *Arch. Surg.*, vol. 75, page 928.

LITWIN, M. S., PANICO, F. G., RUBINO, C., HARKEN, D. E. and MOORE, F. D. (1959), *Ann. Surg.*, vol. 149, page 188.

MELROSE, D. G., DREGER, B., BENTALL, H. H. and BAKER, J. B. E. (1955), *Lancet*, vol. 2, page 21.

—— (1955), *J. Physiol.*, vol. 127, page 51.

MILNES, R. F., WOUDE, R. V. I., SLOAN, H. and MORRIS, J. D. (1958), *A.M.A. Arch. Surg.*, vol. 77, page 13.

SEALY, W. G., BROWN, I. W., YOUNG, G. W., HARRIS, J. S. and MERRITT, D. (1957), *Surg. Gynec. Obstet.*, vol. 104, page 441.

STURTZ, G. S., KIRKLIN, J. W., BURKE, E. C. and POWER, M. H. (1957), *Circulation*, vol. 16, page 988.

WESLOWSKI, S. A., SAUVAGE, L. R. and PINC, R. D. (1955), *Surgery*, vol. 37, page 663.

INTRATHORACIC TRACHEAL RUPTURE AND THE FLAIL CHEST

By HARRY M. WINDSOR AND BRIAN DWYER

Departments of Thoracic Surgery and Anaesthesia, St. Vincent's Hospital, Sydney

THE primary object of this paper is to consider the management of the badly crushed chest. We feel that it is best managed by complete control of respiration.

At St. Vincent's Hospital, Sydney, about 20 moderate to severe chest injuries are admitted each year. An analysis of the mortality and morbidity of 200 such cases in the past eight years leads us to believe that the methods to be outlined in this paper could be used with advantage about three times in every 40 cases. In the past two years they have been used on three occasions.

The decision as to when to use them can be difficult. We feel they ought be used when paradox is so severe that it cannot be controlled by simple means. This is uncommon, for in the 200 cases, although paradox was present in many, it was seldom of greater significance than that found following a thoracoplasty.

Case 1

S. de K., a powerfully built man of 42 years, was involved in a car accident on 16th November, 1957. He was seen by H.W. the next day. He had what could only be described as a "wobbly" chest. All ribs on both sides were fractured, some in two places. He had a small haemothorax, but no pneumothorax, and no significant injury elsewhere. Paradoxical respiration was severe and bilateral. He was extremely dyspnoeic.

The injury was not in itself fatal, but the paradox was uncontrollable by simple means. Unfortunately he was in the Snowy Mountains, 350 miles from Sydney. He was considered too ill to move at the time. This proved to be wrong, for, when two days later he was still alive and the decision to move him was made, he survived a two-hour bumpy trip in a small plane.

He was admitted to St. Vincent's Hospital at 3 p.m. on 19th November. The inevitable had happened. The right lung was wet and opaque. The paradox was extreme.

Respiratory paralysis was at once induced and controlled respiration through a tracheostomy was commenced. It was too late. Adequate ventilation could not be achieved because of widespread consolidation. He died the next day.

Autopsy revealed little other than the comminuted rib fractures with a number of floating segments. Death was due solely to the results of paradoxical respiration. A critical inspection of the many scattered fractures demonstrated how fruitless any endeavour to control the paradox by mechanical fixation, would have been.

This then is the type of case we see about three times in every 40 cases, of moderate to severe chest injury. It illustrates what we mean by severe and dangerous paradox.

In Case 2 the injuries were more extensive and serious. It is reported in detail in order to elaborate the method of control of the paradox and to emphasise the application of modern thoracic surgical and anaesthetic principles to severe chest injuries.

FIG. I. The patient wedged under the dashboard below the broken steering wheel.

Case 2

An American, D. McN., age 44, was involved in a head-on collision at 12.5 a.m. on 19th October, 1958. He received the full impact of the steering column in the chest (Fig. I). He was admitted to Manly Hospital under the care of Mr. Malcolm Stanley and was first seen by H.W. fourteen hours after the accident.

At that time he was lying supine. He was semiconscious, cyanosed and extremely dyspnoeic. There was gross, generalized, surgical emphysema with violent, piston-like, paradoxical movement of the right upper anterior chest and sternal regions. Control of this paradox had been attempted by the use of a hook inserted into an upper right anterior rib. This hook had been attached to a Balkham frame. X-ray of the chest showed a right haemo-pneumothorax which had been treated by a needle in the right pleural cavity. Bronchial obstruction due to secretions was marked.

An abdominal injury was suspected but a satisfactory examination was impossible at this time due to the dyspnoea, the paradox and a confused mental state.

There was a compound fracture of the shaft of the right femur, and a comminuted fracture of the right femoral trochanter. There was a large ragged laceration of the tongue. Most of the front teeth had been knocked out.

The pulse rate was 100 per minute and the respirations 32 per minute. Morphia 96 mgms., pethedine 50 mgms., pethilorfan 100 mgms. and paraldehyde 10 ccs. had been given in an effort to control pain and restlessness.

It appeared to be absolutely necessary to control the paradoxical respiration. This seemed to be possible only by abolishing the patient's spontaneous respiration with a muscle relaxant and instituting intermittent positive pressure respiration. To do this, it was considered essential to transfer the patient, even at great risk, to a Thoracic Surgical Unit.

He was admitted to St. Vincent's Hospital at 4.15 p.m. on 19th October. He was then cyanosed and restless, with frightening paradox and gross bronchial obstruction from secretions.

The management was as follows:—

1. *Bronchoscopy*

   This was done without anaesthesia, and almost proved fatal. The trachea and bronchi were full of blood and solid particles of vomit. The distal light on the bronchoscope rapidly became occluded, blood recollected and the suckers soon blocked. The patient became deeply cyanosed and pulseless.

2. *Intubation with a cuffed endotracheal tube*

   This was done the moment it became obvious that persistence with the bronchoscopy would be fatal. This resulted in more satisfactory ventilation, whilst further removal of intrabronchial blood was possible.

3. *Tracheostomy and insertion of a short cuffed endotracheal tube*

   A large tracheostomy was made and more blood and vomit removed.

4. *Full curarization and controlled respiration*

   Immediately these steps were completed paradox was controlled, but difficulty in maintaining adequate ventilation was noted because of two factors:—

   (a) Increased resistance to inflation

   An X-ray in the theatre showed this to be due to a large right haemo-pneumothorax (the original decompressing needle having blocked). This was decompressed by a large intercostal catheter which drained 20 oz. of blood. The catheter was connected to a suction pump and the lung re expanded. There was no continuous air leak from this pleural cavity.

   (b) Air leak

   An enormous increase in the surgical emphysema developed, and when small incisions were made beneath the clavicles, air hissed out with each squeeze of the bag. It also escaped freely around the tracheostomy so that using a "closed circuit," flow rates up to 10 litres per minute were required to maintain ventilation. Such a leak could only come from a major air passage.

Operation

At 9.15 p.m. a transverse incision was made at the level of the third rib. The moment the subcutaneous tissues were incised, gusts of air obtruded with every squeeze of the bag. The anatomy of the bony injury could now be seen. There was a comminuted fracture of the manubriosternal junction with 2" separation of the ends. There were comminuted fractures of the anterior ends of the upper right ribs. There was complete disruption and 2" separation of the right sterno-clavicular joint. The manubrium was attached to the left clavicle. The mediastinum lay bare.

A rib spreader was inserted. It was an once apparent that the air was issuing from deep in the mediastinum via a dirty discoloured tract. Stomach contents including a few shreds of onion were present in the mediastinum. At this stage there were two difficulties:—

(a) It was almost impossible to keep the lungs inflated because of the large air leak.

(b) Further blood accumulated in the tracheobronchial tree. This increased intrabronchial blood was due to the bleeding from inflamed mediastinal tissues pouring through the air leak.

A large pack was placed in the mediastinum and this resulted in improved anaesthetic conditions. The next difficulty was the finding of the hole. The left innominate vein was stretched taut across the mediastinum and interfered with exposure. It was at once ligated and divided. The innominate artery and aortic arch could then be retracted to the left. The site of the air leak now became apparent. There was a tear one and a half inches long in the trachea. It was situated at the junction of membranous and cartilaginous portions one inch above the bifurcation on the right side. The depth of the wound made closure difficult, but this was achieved with interrupted black silk sutures. It was out of the question to alter the site of the incision to facilitate exposure. At once the anaesthetic problem became less troublesome. It was a little easier to keep the trachea and bronchi dry.

The sternum was wired to the manubrium and the manubrium to clavicle and first rib. This resulted in some degree of stabilization. The wound was closed in layers with one tube in the right pleural cavity and a second in the mediastinum.

Post-operative course

The immediate post-operative period was difficult. Blood, secretions and inhaled vomit were still present and bronchoscopy was again necessary this time through the tracheostomy. It was hurriedly done because the moment assisted respiration was discontinued the patient became deeply cyanosed. However, major secretions were cleared. The cuffed tube was re-inserted and assisted respiration continued.

Immediately following this further difficulty in maintaining adequate ventilation was experienced due to a left pneumothorax. At operation the left pleural cavity did not appear to have been opened, so no tube had been left in that side. This was rectified.

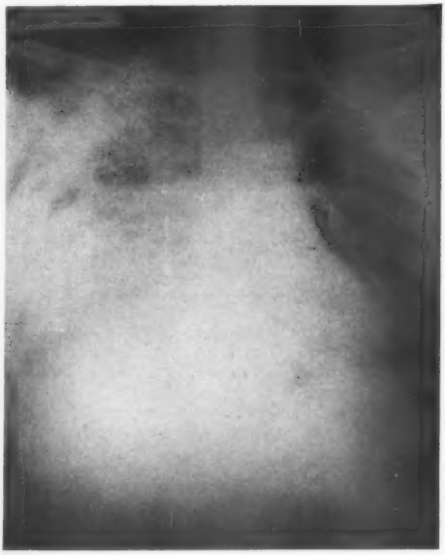


FIG. II. X-ray of chest, immediately following operation.

The problem now was to decide how to maintain adequate respiration. An X-ray taken in the theatre showed both lungs fully expanded though the right lung was extensively consolidated and the left one less so (Fig. II). The trachea had been sutured. the chest wall stabilized so it was anticipated that paradox would be of less significance. In succession the following measures were adopted:—

1. Spontaneous respiration

   The patient was allowed to breathe spontaneously. Seconds showed this was impossible The paradox, despite mechanical fixation, was severe, the breaths were convulsive gasps, bronchospasm was marked and intense cyanosis developed.

2. Assisted respiration

   (a) Manually assisted respiration

   Oxygen (100 per cent.) at a respiratory rate of 40-44 maintained life, but the rapid rate made it impossible to synchronize the rebreathing bag with the patient's respiration. The paradox was unchecked, anoxia and carbon dioxide retention developed.

   (b) Mechanically assisted respiration

   A Bennett's respirator using 100 per cent. oxygen and maximal inspiratory pressures (20-25 cm. of water) was tried unsuccessfully. The secretions, the paradox and the intense bronchospasm provided such a degree of respiratory obstruction that these maximum inflationary pressures were too low and cyanosis, sweating and a raised blood pressure resulted.

3. Controlled respiration

   Complete curarization and intermittent positive pressure respiration proved the only method of controlling the shattering paradox and providing adequate ventilation. The colour improved, the pulse rate dropped to 100 and the blood pressure dropped to within normal limits. Constantly recurring copious secretions and intense bronchospasm persistently caused great concern as the inflationary pressures needed (35 cm. of water) remained very high and rendered two mechanical respirators, the Harrington-James and the Jefferson, ineffectual.

   Manual control of respiration with complete paralysis was essential and was carried out with a standard Boyle's anaesthetic machine and circle type CO_2 absorption. This was continued throughout the night and during the next day, at a rate of 40 per minute at a pressure of 20-35 cm. of water.

First post-operative day

The above measures with frequent tracheobronchial toilet controlled the respiratory difficulties although each bronchial suck out was accompanied by cyanosis, sweating and a rise in blood pressure.

At 6.30 p.m. atropine 1 mgm. and prostigmine 2.5 mgms. were given to reverse the curare and observe any change in respiratory efficiency. Paradox was still severe and both spontaneous respiration and assisted respiration were inadequate.

At 7.30 p.m. the patient's condition had deteriorated with a pulse rate of 150 and a B.P. of 85 mm. of mercury. Controlled respiration was restarted. The blood pressure at once rose to 130 mm. of mercury. Fluctuations in pulse and blood pressure recurred frequently throughout the night. Soda lime had to be changed at fifteen-minute intervals. Pethedine, soluble phenobarbitone and nitrous oxide-oxygen mixture (50 per cent.) sedated the patient.

Second post-operative day

Controlled respiration was continued, but as the curare wore off it was found that respiration could be manually assisted. Despite this in the early afternoon the condition was poor with a pulse rate of 144. The inflation rate was 46 and it was virtually impossible to clear the bronchial tree. A bronchoscopy resulted in some improvement.

At 6 p.m. The curare was reversed and a further attempt at spontaneous respiration made. It was unsuccessful as was assisted respiration. Controlled respiration was induced and maintained throughout the night.

During the period of reversal the first signs of returning consciousness appeared. The patient opened an eye and gazed with a sightless stare. He did, however, respond to painful stimuli.

Third post-operative day

There was further improvement in the conscious state. Manually assisted respiration with smaller intermittent doses of curare was a little easier to maintain. An attempt to induce spontaneous respiration was a little more successful. This time it lasted forty seconds before the patient became distressed. Throughout the night controlled respiration was continued.

FIG. III. X-ray of chest, fourth post-operative day.

Fourth post-operative day

A chest X-ray taken on the fourth post-operative day (Fig. III) shows the extent of the bilateral pneumonia. Throughout the day mechanically assisted respiration with a Bennett's respirator using a 60 per cent. oxygen-air mixture, was alternated with manually assisted respiration. Inflationary pressures were still high (20-25 cm. of mercury). Alevaire and Neoepinine added to the inspired mixture helped to diminish the bronchospasm.

A further attempt at spontaneous respiration was made. There was an improved response, the patient breathing for one minute. Throughout the night to prevent exhaustion and for the sake of safety, respiration was alternately manually controlled or assisted.

This progressive pattern continued for some days. The paradoxical respiration gradually lessened, but severe bronchospasm, secretions and inhalation pneumonia made assisted respiration of some form necessary. Alevaire, Neoepinine, adrenalin in oil, and an intravenous aminophyllin drip were all used and ultimately helped in combating these features. Constant tracheobronchial suction although distressing to the patient was absolutely essential every hour and sometimes more often.

Each day spontaneous respiration continued for a little longer. On the seventh post-operative day it continued for twenty minutes. It was then felt that it was safe to move the patient from the recovery ward and this was done on the eighth post-operative day. The short trip to the room proved most distressing and assisted respiration was necessary for some hours. Throughout the night mechanically assisted respiration was continued. This was the first night that either manually assisted or controlled respiration was unnecessary.

On the ninth post-operative day assisted respiration through a cuffed tube was alternated with spontaneous respiration through a metal tracheotomy tube. At 11 p.m. on this day whilst the metal tube was in place bronchospasm suddenly became severe and secretions copious. This had been initiated by the passage of a duodenal tube a few minutes earlier. The metal tube was removed in order to carry out bronchial toilet. Just as the tube was removed the patient vomited. He inhaled a considerable quantity and at once became unconscious and pulseless. He was sucked out, the cuffed tube was reinserted and manual inflation with 100 per cent. oxygen begun. His pulse returned and his B.P. rose to 250 mm. of mercury.

At no stage had he been without a doctor in attendance. This factor alone saved him on this occasion. Slow improvement took place and three hours later he was asleep and his colour quite reasonable.

On the eleventh post-operative day his condition gradually deteriorated throughout the morning. He had great respiratory difficulty, assisted respiration again becoming necessary. The chest X-ray was worse than ever (Fig. IV). Both pleural cavities were needled and 26 oz. of blood stained fluid removed from the left side. A further bronchoscopy was necessary. The entire bronchial tree was red and dotted with pyogenic membrane. Loose debris was removed. These measures resulted in some improvement and this was reflected in an improved chest X-ray.

Progress was now rapid and on the fourteenth post-operative day he breathed spontaneously for twenty-four hours. The tracheostomy was kept open with a large plastic tube for a further two weeks as the cough was quite inadequate to remove bronchial secretions. He was discharged on 8th January, 1959.

We have deliberately avoided mention of the problems confronting us with regard to the abdominal injury, the femoral fractures and the mediastinal infection.

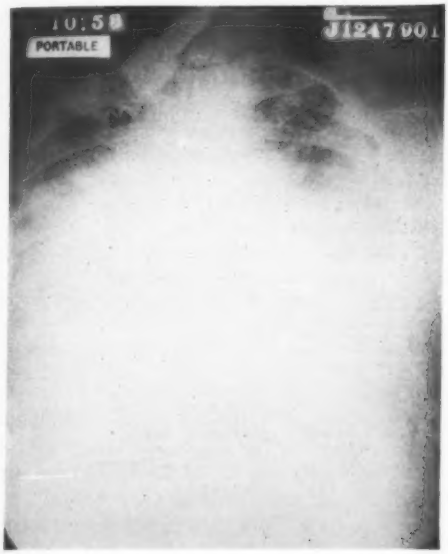


FIG. IV. X-ray of chest, eleventh post-operative day.

COMMENT

Air leak

The air leak is either into the mediastinum and tissues (surgical emphysema), or pleural cavity (pneumothorax).

In the case of a pneumothorax, even if the leak is continuous, management by means of an intercostal catheter and suction pump is a simple matter. If the air leak is severe and continuous management is a different problem for then the assumption must be that:—

(a) the site of the opening is large;
(b) the site of the opening is probably in a major air passage.

Size of the opening

In Case 2 it was not apparent that the air leak was large until such time as an attempt at controlled respiration had been made. The increasing emphysema and the leak of air from the small subclavicular incisions were then obvious with every squeeze of the bag.

Site of leak

Upon the site of the leak will depend the site of appearance of the air. Rupture of the right bronchus, or distal left bronchus both associated, as they are, with the reflection of visceral pleura on to the mediastinum will almost certainly produce either a right or left pneumothorax. There are many reports of such cases. Rupture of the trachea or proximal left bronchus, much more deeply placed will always produce mediastinal emphysema, but not always pneumothorax. Ten reported cases of tracheal rupture have been found in the literature (Table 1). Of these 6 had mediastinal emphysema alone, 2 had mediastinal emphysema and pneumothorax and 2 were doubtful.

The surgical approach of bilateral anterior thoracotomy in Case 2 was decided upon after consideration of the following points:—

1. The ballooning of the sternal area with each inflation suggested the mediastinum as the site of the leak.
2. The absence of a continuous leak of air into the right pleural cavity suggested that the opening was above the right bronchus.
3. It was impossible to carry out adequate tracheo bronchial inspection with a bronchoscope.
4. There appeared to be some possibility of controlling the paradox by fixation of the fragments with wire.
5. Respiration was best controlled and maintained with the patient supine.

Paradoxical respiration

When disruption of the chest wall takes place it leads to paradoxical respiration of a degree proportional to the degree of disruption.

Severe paradox is tantamount to an open sucking pneumothorax; it can be fatal, and is certainly the most difficult of all the mechanical disturbances associated with chest injury to correct.

Most cases can be dealt with by the simple measures of sand bagging and strapping.

Much has been written on the methods of fixation of the thoracic cage by means of wire and various forms of splinting. It is our opinion that if it is feasible to stabilize the paradoxical chest wall with wire, the injury is probably of such a nature that the paradox can be controlled by simple measures. If on the other hand it is impossible to stabilize the chest wall with wire (and this is so in those cases with multiple comminuted rib fractures on both sides with severe bilateral paradox), then it is imperative that stabilization be achieved.

In such a case there is only one way in which this useless form of respiration can be corrected, and that is by the abolition of all repiratory movement and the maintenance of ventilation by intermittent positive pressure respiration. The patient must be rendered apnoeic by some means and respiration taken over by either a respirator or manual compression.

Morch *et alii* did this by hyperventilation with a high capacity respirator. We found it could be simply done with intermittent doses of curare (5-10 mgm. every thirty to forty minutes) and moderate hyperventilation by normal squeezing of the rebreathing bag of a standard anaesthetic machine. We found (despite the wire) that assisted respiration, either manual or mechanical, could not control the paradox and maintain an adequate

TABLE 1

SUMMARY OF CASES OF TRACHEAL RUPTURE

Author	Age	*Sex*	*Cause*	*Clinical Aspects*	*Diagnosis*	*Pathology*	*Treatment*	*Post-Operative*	*Result*
Barford	4	M	—	Surgical emphysema without pneumothorax	—	Left bronchus detached from trachea. Tracheal rupture	Nil		Death
Bickford and Robertson	Young adult	M	Steering Wheel	Surgical emphysema and pneumothorax on the fourth day	On fourth day when tear seen at bronchoscopy	Rupture on the right side at junction of membranous and cartilaginous parts	Right Thoracotomy and closure of the rent	Spasm for thirty mins. Post-operatively before spontaneous respiration	Recovery
Dark and Dewsbury	6	M	Fell 10 feet	Surgical emphysema right pneumothorax	On clinical grounds	—	Right thoracotomy with right upper lobectomy and tracheal repair	—	Recovery
Moty and Guibal		M		Surgical emphysema without pneumothorax		Both bronchi separated from trachea	Nil		Died
Patrick	34	M		Surgical emphysema without pneumothorax		Left bronchus detached from trachea	Nil	—	Died
Richards and Cohn		M	Car accident	Surgical emphysema without pneumothorax	Bronchoscopy and finding a stricture	—	Permanent tracheotomy	—	Alive
Rolleston		M				Rupture of trachea and pulmonary artery	Nil		Died
Schonberg	20	M	Caught between buffers			Trachea transversely severed	Nil		Died
Seuve	74	M	Wheel of bus over chest	Surgical emphysema		Ruptured trachea	Nil		Died
Thompson and Eaton	34	M	Crushed by concrete tile	Right pneumothorax little surgical emphysema	On clinical aspects		Right thoracotomy with right upper lobectomy and repair of trachea		Recovery

minute volume. Similarly in the early stages we found mechanical respirators unsatisfactory. Only manual compression of the bag at 40 per minute with high inspiratory pressures was adequate.

This method provided a far more sensitive control over the respiratory pattern which was constantly changing. Patients with such severe injuries require continuous medical supervision, so this manual control is no great additional burden.

As each day passed paradox diminished and as stability improved and bronchial obstruction lessened it was possible to gain a little co-operation from the patient. He was first introduced to manually assisted respiration, then mechanically assisted respiration (Bennett), using at first 100 per cent. oxygen and then a 40 per cent. mixture in air. Further improvement was noted by the gradual reduction in the inflationary pressures required. Finally assistance to respiration was confined to the nights to enable recovery from the exhaustion of breathing all day.

Sedation

The natural instincts of the medical attendant lean towards the use of sedation to keep the patient unconscious or at least deeply narcotized during such a regime. In this case nitrous oxide and oxygen, pethedine, soluble phenobarbitone, nembutal amytal, chloral and paraldehyde, were all used in varying doses during the first two weeks. We feel that the patient fairly quickly, if subconsciously, adjusted himself to the regime, and most of the drugs merely confused the clinical picture and made assessment difficult without adding a great deal to the patient's comfort. Analgesics alone appeared to be of value.

Light general anaesthesia with nitrous oxide and oxygen was used sparingly and only when all else failed. Bone marrow depression did not occur.

Bronchial obstruction

The most fundamental of the principles of management of a chest injury is that the airway must be clear. It is impossible to describe the enormity of the task in observing this principle in Case 2.

The difficulties were met with at three stages:—

(a) The first six hours

We have outlined and emphasized the difficulties experienced before, during and immediately after operation. Further comment is unnecessary.

(b) The first post-operative week

Throughout this week intrabronchial secretions were excessive. The factors responsible for this were:—

(1) Inhalation pneumonia with a solid right lung and patchy consolidation of the left, due to the initial flooding of the bronchial tree with blood and vomit.

(2) Intense generalized persistent bronchospasm.

(3) Severe purulent tracheo-bronchitis.

It was a full-time task for relays of doctors to attend to this aspect. Suction was carried out with sterile nylon catheters. During the first week the cough reflex was of no value and suction had to be supplemented with the occasional bronchoscopy. Each new man or woman on duty found the task a difficult one and each had to be coached in the correct method of inserting the catheters into both right and left bronchi. Suction was necessary every half-hour and often more frequently. Bronchospasm was ultimately controlled with the help of an aminophyllin drip. The spasm was always accentuated immediately following suction, but this was regarded as inevitable as the secretions had to be removed.

(c) The second and third pos-operative weeks

Further troublesome factors occurred at this stage. They were:—

(1) The inhalation of gastric contents.

(2) The pyogenic membrane in the tracheo-bronchial tree.

(3) The left pleural effusion.

It might appear at first to be a fundamental breach to allow the inhalation of gastric contents. For the first week the patient was maintained on intravenous fluids as the gastric aspirate was copious and offensive. Throughout this period saliva continued to

appear at the tracheostomy, having dropped through the cords and passed through the large tracheal opening above the cuff. This saliva was always carefully removed and the pharynx aspirated before the routine hourly deflation of the cuff on the endotracheal tube.

At the end of a week the patient was able to take sips, but so long as the cuff was inflated most of each sip appeared at the tracheostomy. Each time anything was given by mouth the cuff was deflated, but co-ordination was poor and small amounts always dropped into the trachea. A Ryle's gastric tube was reverted to, but no amount of vigilance prevented the patient from pulling it out, soon after it was inserted. For this reason it was inserted every four hours. It was just after the passage of this at 11 p.m. on the ninth post-operative day that vomiting occurred and a dangerous amount was inhaled.

At a still later stage the crusts from the pyogenic membrane created further difficulties.

It was our object from the start to remove the cuffed tube at the earliest possible moment to prevent pressure necrosis and its sequelae in the trachea. The cuffed tube was finally removed on the fourteenth post-operative day.

Throughout the period of artificial respiration humidification of the inspired air and gases was observed. Alevaire with or without a bronchodilator helped considerably to keep the bronchial secretions moist and mobile.

CONCLUSION

"Not only must the physician be ready to do his duty, but the patient, the attendants, and external circumstances must conduce to the cure."

—HIPPOCRATES.

In the management of Case 2 about 40 doctors were involved. In the first two weeks four "attendants" (an anaesthetist, a resident of senior status, and two sisters) were on continuous duty. This proved a minimum for often more were needed.

If the question of transfer of such patients arises, the risk must be taken. "External circumstances must conduce to the cure." Even then it is not a matter of a spectacular operative save. Rather is it a long, drawn-out struggle demanding the maximum from "physician and attendants" and a "patient" with a fighting heart.

We would like to thank the administration of the hospital, and our colleagues, Mr. Douglas Miller, Mr. Walter McGrath, Mr. Denis Rowe and Dr. Geoff McManis. Their help was spontaneous and valuable.

REFERENCES

AVERY, E. E., MORCH, E. T., BENSON, D. W. (1956), *J. Thorac. Surg.*, vol. 32, page 291.

BARFORD, J. L. (1906), *Lancet*, vol. 2, page 1509.

BICKFORD, B. J. and ROBERTSON, P. W. (1958), *Thorax*, vol. 13, page 246.

DARK, J. and DEWSBURY, P. (1955), *Thorax*, vol. 10, page 62.

MOTY and GUIBAL (1907), *Arch. Méd. Pharm.*, vol. 1, page 444.

PATRICK, J. (1917), *Brit. Med. J.*, vol. 2, page 359.

RICHARDS, V. and COHN, R. B. (1955), *Amer. J. Surg.*, vol. 90 page 253.

ROLLESTON, H. D. (1891), *Trans. Path. Soc. Lond.*, vol. 17, page 42.

SCHONBERG, S. (1912), *Berl. klin. Wschr.*, vol. 49, page 2218.

SEUVE (1873), *Bull. Soc. Anat. Paris*, vol. 48, page 680.

THOMPSON, J. V. and EATON, E. R. (1955), *J. Thorac. Surg.*, vol. 29, page 260.

SURGICAL REPAIR OF POST-OPERATIVE PERINEAL HERNIA

By Anthony R. Kelly

St. Vincent's Hospital, Melbourne

FROM a consideration of the operation, abdomino-perineal excision of the rectum, and the method of perineal repair employed, one would, I think, expect that post-operative perineal hernia would be a common late post-operative complication and that operations for the repair of such herniae would have to be carried out fairly frequently. It is well known however that such is not the case.

Incidence

The incidence of this complication is rather difficult to determine. Gabriel (1945) mentions operating on 3 patients among 500 perineo-abdominal excisions. Hughes (1957) says "rarely the perineal wound becomes the site of an incisional hernia." Cattell (1944) reported repair of such a case; and this was the first case of post-operative perineal hernia requiring operative repair at the Lahey Clinic after 800 abdomino-perineal excisions. Hullsiek (1956) investigated by barium meal studies, 41 patients who had undergone abdomino-perineal excision of the rectum, and estimated that three had perineal herniae, but these were all of a mild degree, and did not need operation. He also reported another case in which repair was carried out. In practice it is found that whilst perineal incisional hernia is not an uncommon complication (probably even commoner that Hullsiek's finding of 3 in 41), it usually consists of only a slight or moderate bulge, and operations for its repair are required extremely rarely.

Pathology

The hernia is of course due primarily to removal of the *levatores ani* muscles and pelvic fascia; and secondarily, to either incomplete closure of the peritoneal floor with stretching of the overlying fibrous tissue of the scar under pressure, or yielding of both the peritoneum and fibrous tissue under pressure. The condition is, as one would expect, commoner in women. The size to which the hernia will enlarge once it becomes established is almost certainly determined by the length of the mesenteric blood vessels and nerves which are the main support of the intestines, and this is particularly the case when the perineal floor is deficient. I consider it is here that we have the explanation as to why, though the condition of post-operative hernia is common, very few patients need operations for its repair. It is because very few patients have mesenteric blood vessels and nerves long enough to allow the intestine to descend sufficiently to produce a large and troublesome hernia. When a large hernia develops, the overlying skin may become very thin, and it may cause the patient considerable discomfort.

Management

Even when a large hernia does develop, surgeons very rarely advise operation. The reasons for this are firstly, because of the operative difficulty of re-creating an adequate pelvic floor, and secondly because the necks of these herniae are almost invariably wide and so the most serious complication of the hernia, strangulation, is most unlikely. Hughes (1957) states "although this is an uncomfortable complication for the patient, special treatment is not required; and in any case, operative intervention might be difficult and prove unsuccessful"; and this is the usual teaching.

In my opinion there are two indications for surgical repair of post-operative perineal hernia. They are, troublesome discomfort for the patient and ulceration of the overlying skin.

Recommended technique for operation

The patient is placed in the prone position on an operating table which "breaks" under the region of the pubic symphysis, so the

head and feet are both low and the perineum is the highest point. First an elipse of stretched skin including the scar is excised. Then the sac wall consisting of peritoneum and overlying fibrous tissue, is cleared of all overlying tissues, opened and any loops of bowel which may be adherent within are freed. The abdomen should be explored through the pelvis to determine whether recurrence or metastases have developed. The first stage of the repair is that of the peritoneum and overlying fibrous tissue. These being redundant, may be "double-breasted" from behind forward. I consider that this repair should not be made at this level of the pelvic brim, as in closure of the pelvic peritoneum after abdomino-perineal excision, but nearer to the original pelvic floor; the suture lines passing from the region of the 5th sacral vertebra forward to the region of the central point of the perineum. Due to descent of the pelvic floor, and stretching of the pelvic peritoneum, the repair can quite conveniently be executed at this level. The next stage in the repair, demands the insertion of some inlay to close the defect in the musculo-fascial layer of the perineal floor. For this purpose, any of the materials used for the repair of large defects in the abdominal wall could possibly be used. My personal preference, based on both theoretical and practical considerations, is for an autogenous graft; and I consider that the most satisfactory substance is fascia lata. Synthetics such as nylon mesh (Parell and Parsons, 1958) have been used. A thick piece of fascia lata of suitable size is removed from the lateral aspect of the thigh and sutured to the firm boundaries of the defect, first to the ligaments overlying the sacrum posteriorly, and to the sacrotuberous ligaments postero-laterally. (As the gluteus maximus muscle takes origin from the superficial aspect of the sacro-tuberous ligament, the graft should be laid on its deep aspect, so that it is united to fibrous tissue, rather than to muscle.) Laterally the graft is sutured on each side to the ischial tuberosity and the inferior ramus of the ischium. The sutures do not actually pass through the bone, but through the falciform process of the sacrotuberous ligament which continues along the bone in this region. Anteriorly the graft is sutured to the posterior edge of the urogenital diaphragm. The placing of the inlay as described closes the defect. The subcutaneous fat and skin are then each closed separately. Post-operative care is carried out along the usual lines. A firm "T" binder is worn for three months.

Report of a case

Mrs. W.F.C., 68 years. This patient was referred to me on 11th June, 1956. She was suffering from a carcinoma of the rectum, situated low in the rectum just above the anal canal. The past history revealed that she had undergone hysterectomy seventeen years previously and anterior colporrhaphy and perineorraphy eight years previously. On 24th July, 1956, an abdomino-perineal excision of the rectum was carried out and, incidentally, a second carcinoma was found in the sigmoid colon. Convalescence was satisfactory and all wounds healed well. The histological report was that both tumours were well differentiated columnar cell adeno-carcinomata. Three lymph nodes associated with the lower tumour, and one associated with the upper tumour were involved with matastatic carcinoma.

On 26th August, 1957, she reported that she was well, and had gained 22 pounds in weight since the operation. She complained of bulging in the perineum, which she had noticed for about six weeks only. She complained of severe discomfort in the region on standing and sitting and she could feel unpleasant movements in the swelling. On examination in the standing position, this position is of course essential in order to assess these cases, a large perineal incisional hernia was found. The dimensions of the swelling were 13 x 13 cm. approximately and I estimated that the fundus of the hernia was about 13 cm. below the level of the site originally occupied by the anus. The overlying skin was very thin and atrophic, and intestinal movements could be plainly seen over most of the area of the surface of the bulge. In the knee chest position the bulge was replaced by a hollow, large enough to hold a tennis ball. On 13th September, 1957, repair of the post-operative perineal hernia was carried out as described above. No metastasis or recurrence was found. Convalescence was uneventful and healing was by first intention.

Review on 1st June, 1959, revealed that there was no obvious hernia or bulge on standing. On coughing there was a small impulse over the perineum. The patient stated that she now had no discomfort in the perineum and she was pleased with the result of the operation. There were no symptoms or signs suggestive of recurrence or metastasis from the original carcinomata.

SUMMARY

Perineal incisional hernia following abdomino-perineal excision of the rectum is

discussed, with special reference to the incidence, pathology, and a recommended technique for surgical repair.

A case of post-operative perineal hernia following abdomino-perineal excision of the rectum, successfully treated by surgical repair, is reported.

REFERENCES

CATTELL, R. B. and CUNNINGHAM, R. M. (1944), *Surg. Clin. N. Amer.*, vol. 24, page 679.

GABRIEL. W. B. (1945), "The Principles and Practice of Rectal Surgery." London, K. H. Lewis & Co. Ltd.

HUGHES, E. S. R. (1957), "Surgery of the Anus, Anal Canal and Rectum." Edinburgh and London, E. & S. Livingstone Ltd.

HULLSIEK, H. E. (1956), *Amer. J. Surg.*, vol. 92, page 735.

JENKINS, J. T. (1946), *Amer. J. Surg.*, vol 71, page 84

PORELL, W. J. and PARSONS, L. (1958), *Surgery*, vol. 43, page 447.

HEALING OF END-IN-END ANASTOMOSES

By A. M. Cuthbertson

Department of Pathology, University of Melbourne

END-IN-END anastomosis is a well recognized surgical manoeuvre for joining hollow tubes where an end to end anastomosis is not readily practicable. Difficulties in attaining mucosal apposition are due to the small size of the tube, such as a ureter anastomosed after division. On occasions it may not be possible, owing to extreme technical difficulties from inadequate exposure, to effect a satisfactory end to end union, for example, restorative excision of rectal carcinomata in the obese male.

Many surgeons deplore this procedure because they fear a stricture will result if mucosa to mucosa apposition is not obtained. No clear account of the changes that take place when a mucosal surface is sutured in this way to the peritoneal or adventitial coat could be found. A series of rats was therefore studied in which end-in-end anastomoses were constructed in the terminal ileum and the sequence of events observd both macroscopically and microscopically. It was realized that infection of some degree was bound to occur but, in the event, this did not greatly obscure or modify the method of healing.

Method

Sprague-Dawley rats weighing approximately 300 g. each were used throughout. Under ether anaesthesia the abdomen was shaved and, with aseptic precautions, opened through a midline incision about 3 cm. in length. The marginal artery was divided at the centre of a vascular arcade 3-4 cm. from the ileo-caecal junction and the bowel divided. Two guy sutures of 4/0 plain catgut were inserted in the proximal end and threaded down the bowel as shown in Fig. I and tied to intussuscept the proximal end. The cut end of the distal stump was then joined to the peritoneal coat of the proximal end by a series of interrupted 4/0 plain catgut sutures. The abdomen was closed by a continuous 0 chromic catgut stitch with Michel clips to approximate the skin. One animal was killed each day up to the 10th day and then at increasing intervals up to the 30th day. The specimens were immediately fixed in 10 per cent. formol saline, photographed macroscopically, and then sectioned at right angles to the line of the anastomosis. The sections were stained with haematoxylin and eosin or Van Gieson's stain.

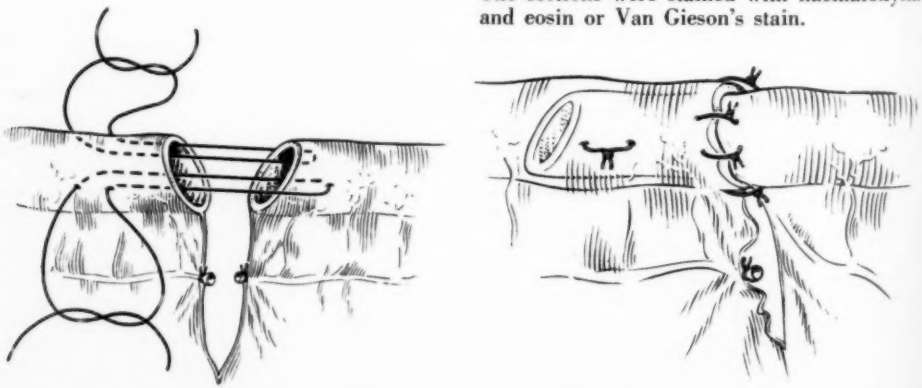


FIG. I. Diagrams illustrating the method used for end-in-end anastomosis in the terminal ileum. Two guy sutures were inserted as shown and tied to intussuscept the proximal into the distal end. The cut end of the distal stump was then joined to the peritoneal coat of the proximal ileum by several interrupted catgut sutures.

RESULTS

Approximately one-third of the animals died between the second to tenth post-operative days. Death was preceded by profuse diarrhoea and at autopsy it was found that the proximal ileum was greatly distended. The cause of death was incomplete bowel obstruction which was due to swelling of the intussuscepted segment of bowel.

Macroscopically the specimens excised from the surviving animals showed changes illustrated in Fig. II. After twenty-four to forty-eight hours the anastomosis was wrapped in omentum and the intussuscepted segment had shortened in length and become swollen and there was an obvious fibrinous inflammation on the peritoneal surface although it was not adherent to the adjacent mucosa. From then on this apposed peritoneal coat which was no longer recognizable as serosa, gradually shortened so that the two mucosal surfaces were gradually brought into apposition (Fig. III). Complete healing was not apparent however until about the twenty-fourth day.

Microscopic examination (Figs. IV and V) confirmed the above findings and showed that epithelial growth accounted for little of the healing; most of the defect being bridged by contraction of granulation tissue.

During the first seven days there was always evidence of some obstruction with quite gross dilatation of the proximal ileum which did not subside until about the twentieth day.

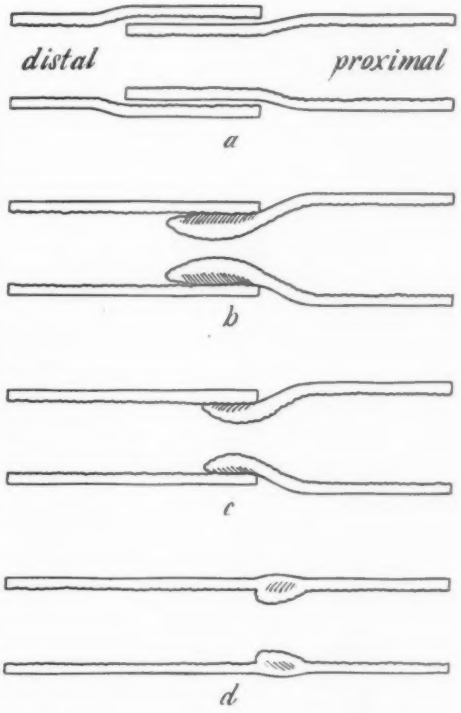


FIG. II. Diagram illustrating the way in which the newly formed connective tissue on the peritoneal coat of the prolapsed segment of bowel contracts so bringing the mucosal surfaces into apposition.

(a) The proximal part of the gut is introduced into and fixed to the distal part;

(b) the end of the proximal segment becomes swollen and gradually shortened;

(c) contraction of this part occurs;

(d) finally, healing is complete, mucosa becoming joined to mucosa.

CONCLUSIONS

1. Following end-in-end anastomosis there is an inflammatory response, both bacterial and traumatic, which is mainly on the peritoneal surface of the intussuscepted portion of the bowel. This inflammation proceeds to granulation tissue formation which contracts at an early stage so that the mucosal surfaces are gradually brought into apposition. Contraction of the longitudinal muscle coat may account for some of the shrinkage that was observed.
2. Growth of the mucosa does not seem to contribute greatly to the re-establishment of mucosal continuity.
3. Bowel obstruction becomes apparent after two to three days but is incomplete and gradually subsides as the intussuscepted portion diminishes in bulk.
4. The final result when only 1 cm. of bowel is intussuscepted is a satisfactory anastomosis for the rat with minimal stricture formation.

DISCUSSION

The way in which healing takes place in an end-in-end anastomosis illustrates the general principles governing the behaviour of connective tissues in healing wounds.

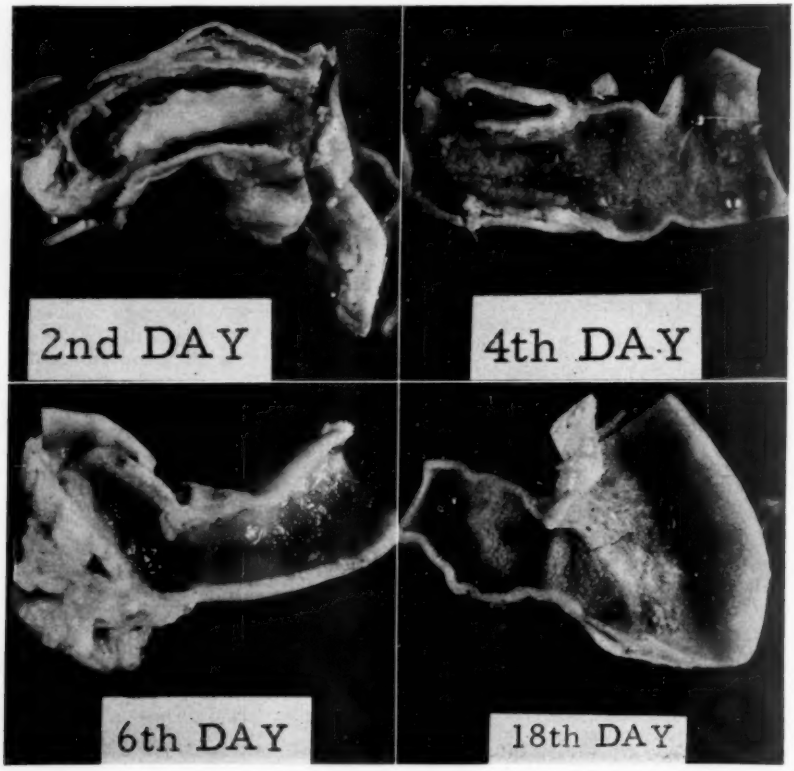


FIG. III. Photographs of specimens of end-in-end anastomosis on the 2nd, 4th, 6th and 18th days. The gradual shortening of the prolapsed segment is well shown with almost total disappearance by the 18th day. Dilatation of the proximal bowel is still apparent on the 18th day.

When connective tissue lies exposed in the base of a skin wound there is invariably some degree of inflammation in the superficial layers which may be due to bacteria, trauma or drying and which usually persists until epithelial cover is obtained. This cellular damage provides the stimulus for the proliferation of the granulation tissue so that progressive deposition of connective tissue results which contains collagen fibre after about the fifth day. As the inflammation subsides in the deeper layers contraction of the connective tissue mass takes place.

The mechanism of this contraction has been fully discussed in a previous paper (Cuthbertson, 1959) and it suffices to say here that it can take place in the absence of collagen fibres. It seems that collagen fibres, although providing the tensile strength of a wound, does not itself undergo active shortening. The ground substance of connective tissue characteristically shows large volume changes with imbibition of water or with dehydration. The fall in capillary permeability as inflammation subsides results in dehydration of ground substance with consequent shrinkage of the tissue. The collagen fibres instead of being arranged in relatively straight parallel bundles become tightly coiled so that in an old stricture the bulk of the tissue is composed of collagen fibres with little ground substance. This process of contraction may continue for some time after the wound has been covered with epithelium so that a stricture may not become apparent until many weeks after the wound has healed.

Partial reversal of this phenomenon may be seen in the dilatation of a tight urethral stricture around an indwelling bougie which is due to an inflammatory response in the stricture with rehydration of the ground substance and consequent partial unwinding of the tightly coiled collagen bundles.

Whether a stricture forms after an anastomosis depends on the time taken for mucosal coverage. Judging from skin wounds, provided mucosal healing is completed within about fourteen days there is little more collagen deposition than in normal tissue and even if narrow at this stage it will soon enlarge under the continuous dilating action of the passing faecal material in much the same manner as the skin grows over a slowly enlarging parotid tumour.

Conversely when mucosal coverage is delayed large quantities of collagen fibre are laid down. It seems that collagen fibre is biologically inert with a slow rate of turnover so that once precipitated it persists for a prolonged period.

The fibre is immensely strong and as the tissue dehydrates the stricture contracts and resists any dilating action of the passing contents.

End-in-end anastomosis provides the conditions conducive to stricture formation but under ideal circumstances with rapid contraction of the intussuscepted bowel healing can be achieved without this unfortunate sequel.

It is realized that the subject of early and late stricture formation is complex and only one aspect has been discussed here. The various methods by which strictures may be avoided and the treatment of the established condition have not been considered here in detail as the particular question under investigation was that of the occurrence of healing of an anastomosis performed by the end-in-end method.

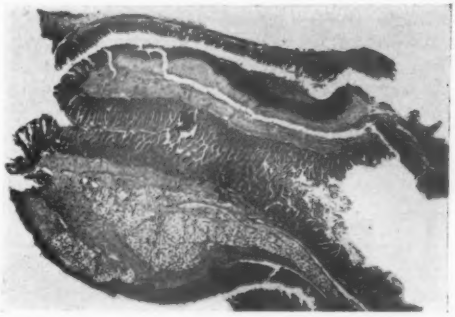


FIG. IV. Low power photomicrograph of a longitudinal section through the prolapsed segment of proximal ileum showing oedema and an intense inflammatory reaction on the peritoneal coat. (x 6)

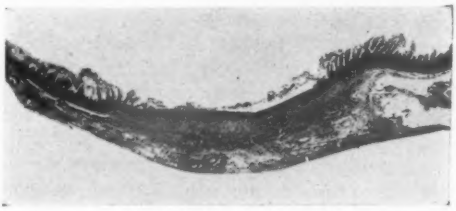


FIG. V. Low power photomicrograph of a section through the anastomosis at 24 days showing the large amount of connective tissue in the base of the wound and the still incomplete mucosal lining. (x 6)

SUMMARY

1. An account is given of the series of changes observed in the healing of an end-in-end anastomosis constructed in the rat's ileum.
2. It was found that mucosal continuity was re-established mainly by the contraction of newly formed granulation tissue on the peritoneal surface of the intussuscepted bowel.
3. Some aspects of stricture formation are discussed.

REFERENCE

CUTHBERTSON, A. M. (1959), *Surg. Gynec. Obstet.*, vol. 108, page 421.

THE CHANGING PROGNOSIS IN APPENDICITIS

By D. S. KIDD

Adelaide

THE place of antibiotics in the control of surgical sepsis has been widely discussed. Attempts have been made to define their value and limitations. In this regard, considerable attention has naturally been given to the commonest surgical suppurative condition in the abdomen, acute appendicitis (Hawke, Becker and Lehman, 1950; Wakeley and Childs, 1950; Rees, 1952; Gilmour and Lowdon, 1952; Harrison, 1953; Erasmus, 1955; Thieme, 1955; Bancroft, 1955). The very commonness of this condition may make it seem beneath the notice of the advanced surgical mind; yet this same commonness makes it, in many ways, a suitable condition for comparison between the pre- and post-antibiotic eras. In particular, appendicitis with perforation has commended itself as an ideal "test condition" for assessing the progress in control of spreading sepsis within the peritoneal cavity.

It was thought, therefore, that it might be of some interest, firstly to survey the trend in appendicitis mortality figures in Australia; and secondly, to correlate these statistics with clinical experience as revealed in the Royal Adelaide Hospital records. In particular, it was hoped that the benefits of antibiotic control could be assessed.

History

To give these figures their correct setting, it is necessary to briefly review the history of appendicitis mortality prior to 1933.

There was a remarkable fall in the death rate from 1904-1912. This was clearly shown by Lett (1914) and McNeill Love (1924) to have been due to patients being brought to operation earlier, with progressively fewer cases in the advanced stages of peritonitis.

TABLE 1

NUMBER OF DEATHS CAUSED FROM APPENDICITIS, AND THE POPULATION IN SOUTH AUSTRALIA AND AUSTRALIA, 1933 TO 1957

Year	*South Australia*			*Australia*		
	Deaths	*Mean Population*	*Deaths per 100,000 Population*	*Deaths*	*Mean Population*	*Deaths per 100,000 Population*
1933	32	581,056	5·5	517	6,630,615	7·8
1934	44	583,343	7·5	546	6,678,349	8·2
1935	40	585,268	6·83	608	6,727,613	9·03
1936	35	587,934	5·95	554	6,780,803	8·17
1937	52	589,663	8·82	552	6,837,590	8·07
1938	39	593,242	6·57	525	6,900,341	7·62
1939	36	597,048	6·03	560	6,968,726	8·03
1940	30	599,136	5·01	450	7,040,661	6·38
1941	26	601,193	4·32	447	7,109,982	6·28
1942	38	609,172	6·24	448	7,176,639	6·24
1943	31	613,327	5·05	384	7,234,651	5·30
1944	35	619,409	5·65	380	7,308,706	5·19
1945	17	627,102	2·71	315	7,389,406	4·26
1946	23	635,127	3·62	338	7,467,474	4·52
1947	33	646,686	5·1	303	7,578,776	3·99
1948	18	661,370	2·71	236	7,709,559	3·06
1949	17	680,287	2·5	194	7,908,890	2·45
1950	13	709,475	1·83	188	8,177,294	2·29
1951	20	732,537	2·73	185	8,420,391	2·19
1952	15	755,042	1·98	174	8,636,657	2·0
1953	9	776,355	1·16	151	8,817,603	1·70
1954	6	796,361	·75	152	8,989,227	1·69
1955	22	820,143	2·68	166	9,202,150	1·80
1956	16	848,531	1·88	155	9,427,291	1·64
1957	11	874,159	1·26	155	9,643,079	1·61

When Officer Brown (1934) surveyed the figures from 1913 to 1933 he showed that there was a gradual but significant rise in the death rate during these years. Thus, for the State of Victoria, the death rate rose from 6.3 deaths per 100,000 population in 1915-19 to 7.6 in 1929-33. This trend was evident not only in Victoria, but also in Queensland, the United Kingdom and the United States of America. The reason for this increased mortality does not seem to have been fully explained.

Fig. I is a graph drawn from the figures for deaths per 1,000,000 population as set

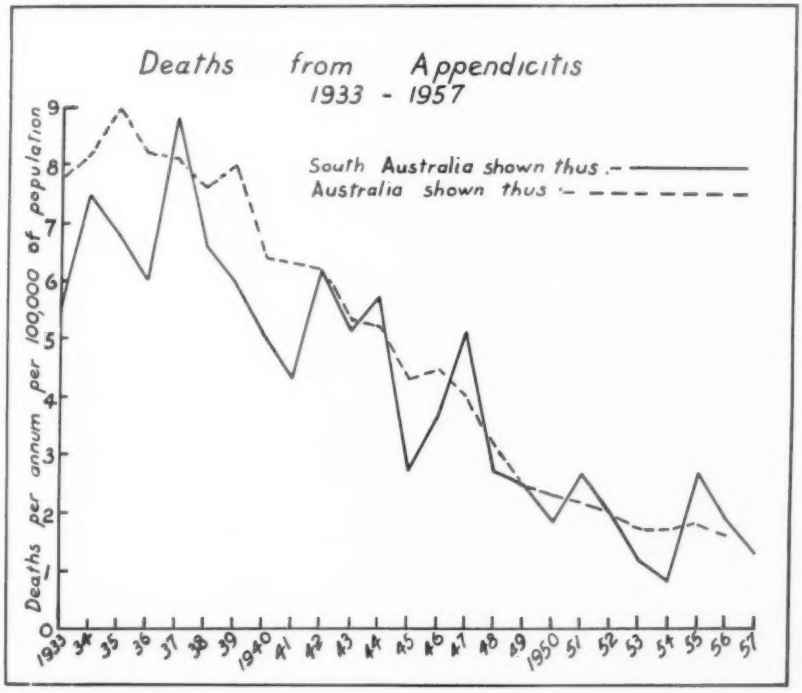


FIG. I

Australian mortality statistics

The first section of this paper then, deals with mortality statistics for appendicitis. These figures were kindly supplied by the Commonwealth Bureau of Census and Statistics.

The overall mortality figures from 1933 to 1957 are set out in Table 1.

It is to be noted that in these figures are included all cases in which the diagnosis of appendicitis was entered on the death certificate. Thus, all post-operative deaths from pulmonary complications, embolism, intestinal obstruction, etc., are included. Moreover, no significant change has occurred, during the period under review, in the classification or method of analysis of the available information. So the figures for the various years should be strictly comparable.

out in Table 1. The South Australian curve shows a few minor fluctuations because of the smaller numbers involved. The curve for Australia runs a fairly level course from 1933-39. Then, from 1940 onwards, there is a progressive downward trend; so that the mortality in 1957 is one-fifth of the figure twenty years previously. This is surely a remarkable achievement. The explanation for this improvement will be considered later.

It is to be noted that this trend has previously brought forth comment from various

Australian authors (Starr, 1940; Kinsella, 1948; Stuckey, 1951). Likewise, a similar trend is in evidence in the Registrar-General's figures for England and Wales (as quoted by Wakeley and Childs, 1950 and Rees, 1952).

various groups is, of course, unknown; therefore case mortality cannot be estimated. However, further reference will be made to the higher mortality in the aged when considering the hospital figures.

TABLE 2

DEATHS FROM APPENDICITIS IN AUSTRALIA ACCORDING TO AGE GROUP

Year	*Age Group*					
	Under 5	*5–14*	*15–59*	*60–69*	*70 and over*	*Total*
1933						
Deaths	25	67	317	70	38	517
Population	568,965	1,252,602	4,133,695	410,298	245,298	6,629,839(a)
Rate per 100,000 pop. in age groups	4·39	5·35	7·67	17·06	15·49	7·80
1938						
Deaths	29	58	331	67	40	525
Population	545,985	1,186,750	4,429,468	436,206	294,674	6,893,083
Rate per 100,000 pop. in age groups	5·31	4·89	7·47	15·36	13·57	7·62
1939						
Deaths	23	64	352	62	59	560
Population	559,710	1,163,521	4,490,471	444,228	302,959	6,960,889
Rate per 100,000 pop. in age groups	4·11	5·50	7·84	13·96	19·47	8·04
1947						
Deaths	16	21	170	43	52	303(b)
Population	760,387	1,138,666	4,746,507	567,183	366,715	7,579,358
Rate per 100,000 pop. in age groups	2·10	1·84	3·58	7·58	14·18	4·00
1955						
Deaths	5	20	66	40	35	166
Population	984,269	1,681,032	5,390,096	690,098	455,196	9,200,691
Rate per 100,000 pop. in age groups	·51	1·19	1·22	5·80	7·69	1·80
1956						
Deaths	7	17	71	27	33	155
Population	1,001,928	1,761,515	5,493,947	700,086	470,082	9,427,558
Rate per 100,000 pop. in age groups	·70	·97	1·29	3·86	7·02	1·64
1957						
Deaths	9	12	58	32	44	155
Population	1,018,957	1,830,461	5,606,083	705,189	482,389	9,643,079
Rate per 100,000 pop. in age groups	·88	·66	1·03	4·54	9·12	1·61

(a) Includes 18,843 ages not stated and not included with age-groups.
(b) Includes 1 age not stated and not included in an age-group.

Mortality in the various age groups

Turning now to an analysis of the figures according to age, Table 2 makes it clear that there has been a reduction in the mortality rate in each and every group. This is well shown in the histogram (Fig. II) which sets out the comparison between the years 1938, 1947 and 1957. Improvement has been most marked in the 15-59 age group, where mortality has been reduced to a seventh; whereas, in the 60-69 age group, reduction is to a third and in the 70 years and over group, to only a half. The incidence of appendicitis in the

Reasons for improving prognosis

Let us now turn our attenton to the possible causes for this remarkable fall in appendicitis mortality. This subject has been discussed at some length by Schullinger, 1947.

It would seem reasonable to suppose that the successive introduction of sulphonamides during the last war, followed in the post-war period by first penicillin, then streptomycin and the broad spectrum antibiotics, has been the predominant factor; supplemented by the more regular use and better understanding

of intravenous infusions and gastric suction, helping to salvage cases in the diffusing peritonitis group. However, several other possibilities suggest themselves:—

1. That less people are developing appendicitis and that therefore the mortality rate is stationary.

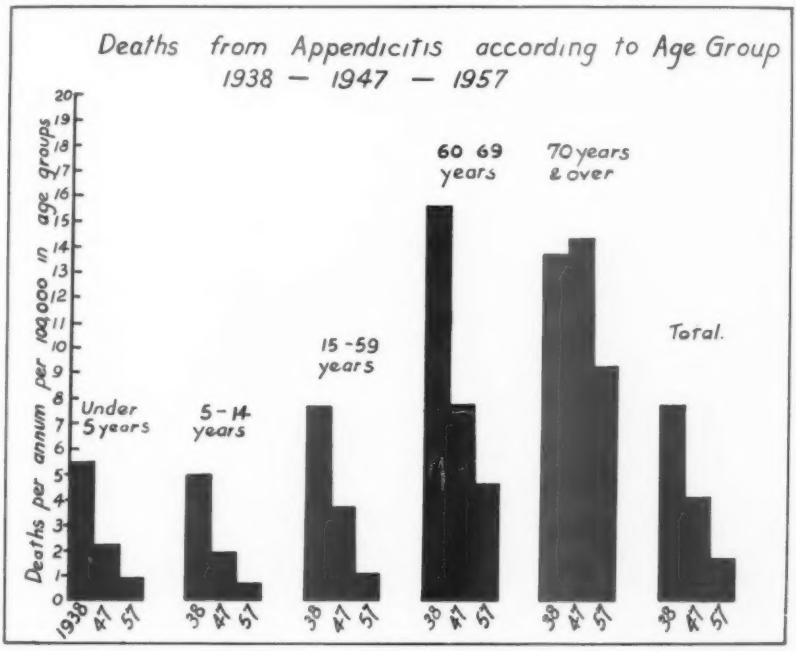


FIG. II

2. That appendicitis has progressively changed its characteristics over the last twenty years, so that, on the average the natural course of the condition is less dangerous. (This possibility has been suggested by Erasmus, 1955.)
3. That the cases are, on the average, being brought to operation earlier with a lower incidence of the more fatal perforative cases.
4. That less anaesthetic deaths are occurring.
5. That some improvement has occurred in the technique of this operation.

The last-mentioned possibility can, of course, readily be dismissed "out of hand." The technique of the operation and presumably the average technical dexterity of the operators, remains unchanged.

It was thought that inspection of the Royal Adelaide Hospital figures might furnish the answer to the other possibilities and the second part of this paper is devoted to these considerations.

Royal Adelaide Hospital series — method of study

In view of the great mass of clinical material available, it was decided to take samplings of cases from pre-war and recent years for comparison. An arbitrary selection was made of all cases diagnosed as acute appendicitis for the years 1936-38. These totalled 786 cases and are hereafter referred to as Series A. For comparison, 786 consecutive cases from the year 1956-7 were selected, hereafter referred to as Series B.

Difficulty was immediately experienced in deciding which cases to accept as bona fide acute appendicitis. If all excised specimens had been subjected to histological examination all would have been simple. But in fact, in Series A (1936-8) only four cases were

recorded as pathologically confirmed. A further 60 cases were described as gangrenous, thus establishing their bona fides. These 64 cases comprised only 9 per cent. of the total. The remaining 91 per cent. had to be accepted on the unreliable basis of macroscopic appearance; and there is no doubt that quite a percentage were not genuine acute appendicitis at all.

TABLE 3

DEATHS FROM APPENDICITIS — ROYAL ADELAIDE HOSPITAL

Classification	*Series "A" (1936–8)*			*Series "B" (1956–7)*		
	No.	*Deaths*	*Percentage*	*No.*	*Deaths*	*Percentage*
Appendicitis	721	6	0·8	670	3	0·4
Appendicitis with perforation	33	9	27·3	85	5	5·9
Appendiceal abscess	31	1	3·2	31	1	3·2
Total	786	16	2	786	9	1·1

The position in Series B (1956-7) was rather better. Here 349 cases were confirmed histologically. A further 27 were described as gangrenous, making a total of 56 per cent. reliably confirmed.

This leaves me in the unsatisfactory position of presenting a comparison between two series which are not strictly comparable. However, the scales are tipped unfavourably against Series B, from which so many of the normal cases had been weeded out by the pathologist; so that any improvement which shows up can be taken as somewhat of an underestimate.

On the other hand, it is reasonable to accept as comparable the cases listed as appendicitis with perforation in the two series.

Mortality figures — Royal Adelaide Hospital

Table 3 summarizes the comparisons between the two series. Several features are worth noting:—

1. The higher incidence of appendicitis with perforation in Series B (roughly 2½ times Series A). I think this is largely due to the higher percentage of genuine acute appendicitis in this series. At least, this observation seems to dispose of two of the above suggestions; first, that appendicitis is becoming a milder disease and second, that cases are being brought to operation earlier.
2. It is interesting that, in these strictly comparable groups of perforative appendicitis, the mortality rate has been reduced from 27.3 per cent. to 5.9 per cent., i.e. roughly to one-fifth, which you will recall is the same as the reduction shown in the Commonwealth Mortality Statistics. It is in this group that the greatest improvement has naturally enough shown up.
3. It is to be noted that all but five of the perforated appendicitis cases in Series B were given antibiotics, mostly penicillin and streptomycin, but sometimes one of the broad-spectrum antibiotics. The five cases not given antibiotics survived without complication.
4. Two hundred of the 670 simple appendicitis cases in Series B were given antibiotics. Mostly this was a prophylactic

TABLE 4

PERCENTAGE DEATHS FROM APPENDICITIS (W. S. REES' FIGURES)

Classification	1934–7	1940–3	1947–50
	Percentage	*Percentage*	*Percentage*
Simple acute appendicitis	0·4	0·4	0·3
Appendicitis with localized peritonitis	3·5	2·8	0·7
Appendicitis with generalized peritonitis	36·0	27·0	12·0
Appendiceal abscess	10·8	6·1	7·9
Totals	5·4	4·3	2·3

measure but, often these drugs were reserved for cases showing complications. The mortality of 0.4 per cent. in this series probably represents the irreducible minimum. There was not a single death in 1957; in 1956 the deaths were three — one from a combination of haematemesis and myocardial ischaemia, one death under anaesthesia and one from pulmonary embolism.

5. It is suspected that the post-war improvements in anaesthesia may have contributed to the lowered Commonwealth mortality figures; but this factor does not show up in the small hospital series.

For comparison Table 4 shows the figures presented by Rees (1952) and Table 5 by Thieme (1955). In general, the same trends are apparent, though it is interesting to note the higher mortality for appendiceal abscess in the earlier years in these series, as compared with the Adelaide figures.

TABLE 5

DEATHS FROM APPENDICITIS — E. T. THIEME (ANN ARBOR)

Classification	1935–39			1940–44			1947–51		
	Cases	*Deaths*	*Percentage*	*Cases*	*Deaths*	*Percentage*	*Cases*	*Deaths*	*Percentage*
Simple acute appendicitis	308	1	0·32	463	0	0	353	0	0
Appendicitis with localized peritonitis	77	4	5·2	112	4	3·3	127	1	0·7
Appendicitis with generalized peritonitis	9	2	22	18	3	14	9	1	11
Appendiceal abscess	23	4	17·3	28	0	0	15	0	0
Total	417	11	2·6	622	7	1·1	504	2	0·4

Causes of death

Table 6 sets out the causes of death in the two series. Most of the deaths in Series A were due to general peritonitis; most of the deaths in Series B were due to extra-abdominal factors, such as cardiac or lung complications. (In one case of this series, it was not possible to decide from the autopsy report whether small bowel obstruction on the one hand or bronchopneumonia and left ventricular failure on the other hand was responsible for the death. Hence the half-and-half rating.)

Appendicitis in the aged

The figures available in the present hospital survey are hardly sufficient to be

TABLE 6

CAUSES OF DEATHS IN ROYAL ADELAIDE HOSPITAL SERIES

Cause of Death	*Series A* 1936–8		*Series B* 1956–7	
	Appendicitis	*Appendicitis with perforation*	*Appendicitis*	*Appendicitis with perforation*
Abdominal				
General peritonitis	4	9		1
Small bowel obstruction				½
General				
Coronary thrombosis	1			
Pulmonary embolism			1	
Death from anaesthesia	1	0	1	1
Myocardial ischaemia } Haematemesis }			1	
Acute pulmonary oedema				1
Bronchopneumonia				1
Bronchopneumonia and left ventricular failure				½

statistically significant. Thus, only 18 cases in Series A were over 60 years (i.e. 2.3 per cent.) compared with 69 cases in Series B (i.e. 8.8 per cent.). The higher proportion in the second series may be explained, in part by the ageing population and in part by the higher proportion of younger people able to afford private hospital treatment.

The death rate for over 60 years of age was 28 per cent. in Series A and 5.8 per cent. in Series B. Table 7 shows that these death rates are in line with the collected figures recorded in a recent article by Christensen (1958).

TABLE 7

MORTALITY RATE FOR APPENDICITIS IN THE AGED

(Modified from Christensen)

	No.	*Mortality (Percentage)*
Fitch (1928)	13	54
Lewin (1931)	43	28
Wood (1934)		28
Taylor (1935)		20
Royal Adelaide Hospital (1936–38)	18	28
Stalker (1940)	82	15·9
Simpson (1946)		25
Scheibel and Moise (1949)	53	13
Wolff and Hindmann (1952)	88	4·5
Christensen (1955)	90	5·6
Royal Adelaide Hospital (1955–57)	69	5·8

Comparison with the mortality for cases under 60 years of age of 1.4 per cent. in Series A and 0.7 per cent. in Series B shows how much graver the prognosis remains in the aged. Factors responsible for this may be summarized as follows (Aird, 1957; Christensen, 1958; Carp and Arminio, 1952; Wolff and Hindmann, 1952):—

1. Difficulties of diagnosis in the aged.
   These difficulties are due to
   (a) Deceptively mild symptoms and signs in the aged. Patients tend to be poor historians, being vague and subject to memory defects. They are often casual and stubborn about all illnesses. Owing to dulled sense of pain the onset and progress of the disease tends to be insidious, so that their pains are difficult to evaluate. As Rolleston (1932) (quoted by Christensen, 1958) neatly puts it, "The organs suffer in silence, without any local or general disturbance."
   (b) The frequent presence of underlying degenerative diseases (e.g. cerebral arterial disease, Parkinson's disease) masks the clinical picture.
   (c) The relative infrequency of the condition in the aged, causing it to be overlooked in the differential diagnosis.
   (d) The difficulty of differentiation from other abdominal conditions.
2. Greater danger of abdominal disease in the aged, with more rapid progress to perforation and peritonitis.
   (a) Brotherus (1955) suggested this may be due to thickened vessels, leading to vascular occlusion, with earlier onset of gangrenous perforation.
   (b) Carp and Arminio (1952) attribute it to lessened resistance to infection owing to the atrophy or complete lack of lymphoid tissue follicles in the appendix in the aged.
   (c) The well-known tendency for older people to regard purgation as a

TABLE 8

COMPARISON BETWEEN 69 PATIENTS AGED 60 OR MORE, AND 717 UNDER 60 YEARS (1956-7)

	Under 60 years	*Over 60 years*
Acute appendicitis	625 = 87·2 per cent.	45 = 65·1 per cent.
Appendicitis with perforation	70 = 9·7 per cent. } 12·8 per cent.	15 = 21·7 per cent. } 34·9 per cent.
Appendiceal abscess	22 = 3·1 per cent. }	9 = 13·2 per cent. }

panacea for all ills, may well be a further factor contributing to the early onset of perforation.

3. Increased pulmonary and cardiovascular complications.

Table 8 clearly shows the higher incidence of perforation in the older age group. Thus the combined figures for appendicitis with perforation and appendiceal abscess comprise 34.9 per cent. of the total for the over 60 years group and only 12.8 per cent. in the under 60 years group.

TABLE 9

COMPLICATIONS APPENDICITIS (SURVIVING CASES)

	1936–8 (715 *Cases*)	1956–7 (667 *Cases*)
Abdominal		
Wound sepsis and abscess	13	22
Ileus	1	10
Pelvic abscess	3	2
Drained through wound	1	
Drained surgically per rectum	2	2
Mechanical obstruction	1	
Small bowel strangulation	1	
Burst abdomen	1	
Faecal fistula	1	1
"Spastic ileus" with transverse colostomy		1
Acute retention		1
Other Complications		
Bronchitis and Pneumonia	10	9
Atelectasis	1	3
Deep venous thrombosis	2	2
Superficial thrombophlebitis	1	
Pulmonary embolus	2	
Congestive cardiac failure		2
Large sacral bedsore		1

Post-operative complications

Finally, an attempt was made to survey the post-operative complications in the two series.

Table 9 sets out these complications for all surviving cases of appendicitis.

Here the inadequacy of the records is even more apparent, particularly in the pre-war series. For instance, the figures for wound sepsis and ileus can be dismissed as of no significance. Clearly listing of these complications in the records depends firstly on what one calls wound sepsis and where meteorism is thought to end and ileus to begin; and secondly on the completeness of the records.

The most that one can conclude is that the post-operative course of surviving cases was not much more eventful in the pre-antibiotic era; and interestingly, that there seems to have been little change in the incidence of chest complications in the two series.

Table 10 sets out the comparison between the surviving cases of appendicitis with perforation in the two series. The post-operative course seems to have been extraordinarily uneventful in both series, apart from ileus and wound sepsis, the figures for both of which are probably significant only in Series B.

TABLE 10

COMPLICATIONS APPENDICITIS WITH PERFORATION (SURVIVING CASES)

	1936–8 (24 *Cases*)	1956–7 *(80 *Cases*)
Abdominal		
Wound sepsis	2	11
Secondary haemorrhage (from wound)	1	
Pelvic abscess		
Drained per rectum	2	2
Mechanical obstruction	2	
Ileus		6
General		
Pneumonia	1	2
Scrotal abscess		1
		*(Antibiotics in all except five cases.)

DISCUSSION

1. It may be concluded, then, that the greatly improved prognosis in appendicitis is in no way attributable to any change in the character of the disease; nor to any outstanding improvement in earlier diagnosis or in surgical technique; but that the introduction of antibiotics, supplemented by the more adequate use of drip and suction has been largely responsible.

It must be stressed, however, that antibiotic therapy is not all-powerful and is to be regarded as "supplemental, rather than substitutional" (Carp and Arminio, 1952). There remains a pressing need to get patients to operation earlier and to observe all possible refinements of pre- and post-operative care to reduce the mortality to the irreducible minimum.

2. Garlock (1947) emphasized the risk of antibiotics masking the picture of the acute abdomen if used pre-operatively, thus "lulling the physician and patient into a false sense of security."

Other writers (e.g. Meyer, 1949) warn against the risk of the masking of post-operative complications amongst those patients receiving heavy antibiotic therapy (e.g. Meyer cites a case of insidious left subphrenic abscess rupturing into the pericardium, with a rapidly fatal outcome).

No evidence of these risks showed up in the examination of the Royal Adelaide Hospital case records. While it is as well to keep these possibilities in mind, no such considerations should be allowed to obscure the great benefits of antibiotics, especially in the presence of peritonitis.

3. In the past, much discussion has taken place over the relative virtues of conservative versus operative management of appendicitis in its various stages. (See Hawke, Becker and Lehman, 1950, for a review of the literature together with a comparison of their results using the two types of management.)

While there still remains some difference of opinion, it seems to be now widely accepted that modern ancillary aids have so increased the safety of operative measures that the place for conservative management is now limited to those cases of several days' duration in which there is a sharply localized firm mass; and that even in these cases, in the absence of prompt improvement from the Oschner-Sherren regime, there should be no hesitation in proceeding to drain the abscess (removing the appendix at the same time only if it is reasonably accessible). Cases of perforation with diffuse peritonitis should be submitted to immediate appendicectomy (unless diagnosis has been so long delayed that the patient is in extremis when first seen by the surgeon).

It seems that in these days of satellites and projected lunar expeditions marvels are so commonplace that we hesitate to enthuse any more over advances in medicine. But surely it is reasonable to feel some satisfaction and even pride in seeing the mortality of this common condition reduced in twenty years to a fifth; which means that in 1957 alone, over 600 Australians, mostly in the prime of life, lived through an attack of appendicitis which, under the conditions prevailing twenty years ago, would have proved fatal.

SUMMARY

1. The history of appendicitis mortality prior to 1933 is briefly reviewed.
2. Mortality statistics for appendicitis in Australia from 1933 to 1957 are presented, both for the population as a whole, and for the various age groups. A remarkable reduction in mortality rates is in evidence.
3. Possible reasons for the improving prognosis are advanced. These possibilities are examined in the light of two series of appendicitis cases from the Royal Adelaide Hospital (Series A from 1936-8, Series B from 1956-7). Antibiotics, intestinal decompression and intravenous infusions appear to have been the main factors contributing to the improved prognosis. This improvement has occurred principally in the group of perforative-appendicitis.
4. The graver prognosis of appendicitis in the aged is shown; and factors responsible for this are summarized. This older group remains a challenge for further improvement.
5. Post-operative complications in the Royal Adelaide Hospital Series are reviewed.

ACKNOWLEDGEMENTS

I wish to thank Mr. A. C. McEachern for his helpful suggestions and constant encouragement in the preparation of this paper.

The ready help given by the Deputy Commonwealth Statistician and Government Statist, in supplying the required statistical information, is also gratefully acknowledged.

REFERENCES

AIRD, I. (1957), "A Companion in Surgical Studies," Second Edition. Edinburgh and London, E. & S. Livingstone Ltd.

BANCROFT, F. W. (1955), *Surg. Clin. N. Amer.*, vol. 35, page 411.

BROTHERUS, V. (1955), *Nord. Med.*, vol. 53, page 543. (Cited by Christensen, 1958.)

BROWN, C. T. Officer (1934), *Med. J. Aust.*, vol. 2, page 407.

CARP, L. and ARMINIO, J. A. (1952), *Amer. J. Surg.*, vol. 83, page 773.

CHRISTENSEN, E. (1958), *Brit. Med. J.*, No. 2, page 832.

ERASMUS, J. F. P. (1955), *Med. J. S. Afr.*, vol. 29, page 78.

GARLOCK, J. H. (1947), *New York J. Med.*, vol. 47, page 1604.

GILMOUR, I. E. W. and LOWDON, A. G. R. (1952), *Edin. Med. J.*, vol. 59, page 361.

HARRISON, P. (1953), *Amer. J. Surg.*, vol. 85, page 160.

HAWK, J. C., BECKER, W. F. and LEHMAN, E. P. (1950), *Ann. Surg.*, vol. 132, page 729.

KINSELLA, V. J. (1948), *Med. J. Aust.*, vol. 1, page 544.

LETT, H. (1914), *Lancet*, vol. 1, page 295.

LOVE, J. MCNEILL, (1924), *Brit. J. Surg.*, vol. 12, page 232.

MEYER, N. C. (1949), *Ann. Surg.*, vol. 129, page 148.

REES, W. S. (1952), *Lancet*, vol. 2, page 1199.

ROLLESTON, H. (1932), "Medical Aspects of Old Age," page 156. London, Macmillan. (Cited by Christensen, 1958).

SCHULLINGER, R. N. (1947), *Ann. Surg.*, vol. 126, page 448.

STARR, K. W. (1940), *Med. J. Aust.*, vol. 1, page 71.

STUCKEY, E. S. (1951), *Med. J. Aust.*, vol. 2, page 804.

THIEME. E. T. (1955), *A.M.A. Arch. Surg.*, vol. 70, page 207.

WAKELEY, C. P. C. and CHILDS, P. (1950), *Brit. Med. J.*, vol. 2, page 1347.

WOLFF, W. and HINDMANN, R. (1952), *Surg. Gynec. Obstet.*, vol. 94, page 239.

POST-OPERATIVE ADRENAL INSUFFICIENCY

By Patricia Wilson

Melbourne

Introduction

UNTIL recent years acute post-operative adreno-cortical insufficiency was a relative rarity; frequently not diagnosed before death and, when recognized, treated inadequately. It is only in the past eight years that the importance of the adrenal cortex in the body's response to stress, including the stress of anaesthesia, major and minor surgery, has been evaluated.

The introduction of adreno-cortical hormones has made possible adequate replacement therapy and management of the adrenal crisis. On the other hand, the extensive use of large doses of these hormones in a wide variety of medical and surgical conditions has been responsible for the marked increase in incidence of this complication. The purpose of this paper is to draw attention to the very real dangers of acute adreno-cortical insufficiency in certain types of surgical patient, and to indicate the prevention and management of this complication.

Causes of Adrenal Insufficiency in Surgical Patients

I. Primary adrenal hypofunction.
   (a) Adrenal disease.

II. Secondary adrenal hypofunction.
   (a) Pituitary hypofunction.
   (b) Drug induced.

III. Surgical removal of adrenal tissue.

IV. Non-specific causes of adrenal insufficiency.
   (a) Toxaemia.
   (b) Repeated stress

Signs of Acute Adrenal Insufficiency

The typical picture of acute adreno-cortical insufficiency is that of sudden and extreme circulatory collapse which does not respond to the usual methods of resuscitation, including transfusion of blood and the administration of pressor drugs. Frequently, despite the low blood pressure, the patient appears well and mentally alert. Later mental confusion, coma and hyperpyrexia may also be apparent.

The circulatory collapse is usually very sudden and the untreated patient may die within half to one hour. Therefore prompt treatment is essential and even if there is some doubt about the diagnosis, administration of adreno-cortical hormones can do no harm and may be lifesaving.

Biochemical studies show that intense adreno-cortical stimulation commences during or shortly after surgery and continues for one to three days (Hume, 1953; Franksson *et alii*, 1954; Tyler *et alii*, 1952). The degree of stimulation appears to depend on the extent of the surgical trauma, blood loss and duration of operation. The type and duration of anaesthesia appear to play a relatively minor role. Hence, the surgical patient who has actual or potential adrenal cortical inadequacy is most likely to develop acute adrenal insufficiency in the post-operative stage although occasionally it may appear during operation. Any post-operative complications such as infection or haemorrhage also tend to precipitate adrenal insufficiency.

The Management of Acute Adrenal Insufficiency

As the increase in incidence of this complication is almost entirely due to the enormous increase in use of cortisone and allied compounds in therapeutics, the main part of this paper will be devoted to a discussion of the management of this type of case. Other groups will be mentioned briefly.

(i) *Primary adrenal hypofunction*

This is present in Addison's disease and in the Waterhouse-Friderickson syndrome where there is haemorrhage into the adrenal gland, associated with septicaemia.

The management of such patients for surgery involves the administration of increased doses of cortisone pre-operatively, and intravenous cortisone during and after operation.

A drop in blood pressure at any stage must be counteracted by immediate infusions with cortisone or hydrocortisone and pressor agents. Surgery in these patients is, at the best, a hazardous procedure.

(ii) *Surgical removal of adrenal tissue*

Bilateral adrenalectomy for generalized malignancy is not uncommon. It is obvious replacement therapy must be instituted — this is best commenced before the removal of the first adrenal (Latham, 1956).

Tumours of the adrenal cortex which result in symptoms of hypercorticism often cause atrophy of the contralateral adrenal gland. Hence when such tumours are removed the patient must be treated with large doses of cortisone before and after operation until such time as the remaining adrenal resumes normal function.

(iii) *Secondary adrenal hypofunction*

Hypofunction of the anterior pituitary gland will result in absence of or decrease in adreno-corticotrophin which is the normal stimulus of the adrenal cortex. This may occur with atrophy, fibrosis or tumours of the pituitary, in brain injuries and in surgical hypophysectomy. These cases are just as likely to collapse with the stress of surgery as cases of primary adreno-hypofunction and must receive adequate replacement therapy prior to surgery.

DRUG INDUCED ADRENO-CORTICAL INSUFFICIENCY

Cortisone and allied compounds are employed in a large variety of unrelated medical conditions. In contradistinction to their use in replacement therapy, large doses must often be employed in alleviating medical conditions and therefore side effects are more likely to occur. Most important of these side effects is hypoplasia of the adrenal glands and suppression of adrenal function. This hypofunction may be compensated until such time as stress such as surgery is applied when acute adrenal insufficiency may occur.

This adrenal suppression is more likely to occur if the dosage of cortisone is high and of long duration. However, adrenal atrophy has been reported after as little as five days' treatment with cortisone and acute insufficiency has developed in a patient twenty-four months after the cessation of cortisone (Salassa *et-alii*, 1953). Minor procedures such as manipulation of a joint have been reported as precipitating fatal adrenal insufficiency (Harnagel and Kramer, 1955), although more commonly, and certainly in my experience, the complication has appeared after major abdominal operations.

In fatal cases autopsy shows the adrenal glands to be atrophied, usually with marked thinning of the cortex and ballooning of the cortical cells. Frequently this is the only demonstrable abnormality at autopsy.

In view of the ever-increasing number of reports of this complication, particularly in American literature where cortisone has been employed since 1949 (England since 1955), I now use a modified but much more vigorous dosage of cortisone than I suggested when first writing on this subject in 1956 (Wilson, 1956). Since then I have been concerned in the management of 93 major and 41 minor cases where cortisone has been involved. Six of these major cases have died from adrenal insufficiency even though the dangers were appreciated and the patient on a high dosage.

The scheme of management for all patients on cortisone, ACTH or allied drugs is as follows:—

(i) A history of cortisone administration is obtained, if possible. It must always be suspected in patients with:

Rheumatoid conditions.
Severe bronchial asthma.
Lupus erythematosis.
Ulcerative colitis.
Skin conditions.
Blood dyscrasias.
Eye conditions.
Nephrosis.

The common major surgical conditions where such patients are encountered are:—

Ulcerative colits for colectomy.

Thrombocytopaenic purpura and haemolytic anaemia for splenectomy.

Gastric ulcer for gastrectomy.

(ii) A history or the presence of signs of hypercortisonism should be elicited. If present there is almost certainly also adrenal suppression. Cushing-like effects — obesity of the trunk and "mooning" of the face are

the common physical signs. Sodium retention with oedema, hypertension and psychic changes may also occur.

(iii) Tests of adrenal function are not of great value because they are unreliable and time consuming and do not necessarily foretell how the adrenal will react under surgical stress.

OPERATIVE MANAGEMENT OF CASES WITH SUSPECTED ADRENAL SUPPRESSION

There are 3 classes of patient.

1. The patient with a history of brief cortisone administration some time previously and for a minor operation.

This patient is probably quite safe without any cortisone coverage but must be watched carefully after operation and intravenous cortisone given if the blood pressure falls.

2. The patient with a history of prolonged administration or high dosage for minor operation.

Cortisone should be commenced the day before operation — 100 mgm. orally morning and night. Cortisone 100 mgm. is given intramuscularly two hours pre-operatively and is continued in gradually decreasing dosage, preferably orally, for five days after operation.

If the patient is already on cortisone, the maintenance dose should be doubled or tripled. If, at any time blood pressure falls and hemorrhage is excluded, intravenous cortisone must be given.

3. The patient for a major surgical operation.

This section includes major abdominal surgery or any operation where much trauma or blood loss is expected. In my experience it is in this group that death can readily occur unless constant vigilance is maintained both during and for seven days after operation.

The anaesthetic must be administered with great care with no hypoxic episodes, surgical trauma must be at a minimum and blood loss immediately replaced. At no stage during or after the operation should hypotension be allowed to persist.

Cortisone dosage is as follows:—

(a) 100 mgm. b.d. one day pre-operatively or double the dosage if the patient is already on cortisone.

(b) Intravenous cortisone drip commenced one hour pre-operatively and a minimum of 200 mgm. given up to the end of the operation. Post-operatively continuous intravenous cortisone 100 mgm. six hourly for twenty-four to forty-eight hours is administered followed by intermittent intravenous cortisone hemisuccinate for a further forty-eight hours — thereafter intramuscular or oral cortisone is given according to the condition of the patient.

This regime varies slightly with the patient and the type of operation, previous dosage, etc. If any post-operative complication such as sepsis, haemorrhage supervenes the dose must be increased. Hypotension must be treated immediately with intravenous cortisone.

Careful watch must be maintained over intravenous fluids especially while the continuous cortisone is being employed. Sodium and potassium balance must also be watched.

Sepsis, shock and haemorrhage may be masked because the patient on this high dosage has a marked euphoria and absence of physical signs — hence very careful observation is necessary.

COMPLICATIONS OF HIGH CORTISONE DOSAGE IN SURGICAL PATIENTS

There is an increase in operative and post-operative complications in patients on high dosage although in the majority of cases these are minor compared with the hazards of acute adrenal insufficiency.

(a) Tissues tend to be oedematous and there is more marked capillary oozing during operation.

(b) Sepsis and even septicaemia is more likely to develop with minimal signs.

(c) Masking of shock and haemorrhage.

(d) Perforation of a viscus with minimal signs and symptoms.

(e) Overloading with fluids, sodium retention and potassium depletion.

(f) Impairment in wound healing — this is a much over-rated complication. The possibility of delayed healing has in the past been responsible for the pre-operative withdrawal of cortisone by surgeons and physicians, resulting in unnecessary post-operative deaths.

Of the 134 cases with which I have been associated, wound breakdown has occcurred in 5 patients; all major abdominal cases with established peritonitis.

(g) Psychosis. This complication has been encountered in the series in one patient in the post-operative period.

NON-SPECIFIC CAUSES OF ADRENAL INSUFFICIENCY

Evidence is accumulating that in some cases not fitting into the above groups, the appearance of intractable hypotension during or after surgery and anaesthesia and delay in recovery from anaesthesia may be a manifestation of failure of the adrenal cortex (Galante *et alii*, 1954; Howland *et alii*, 1956).

Such cases include—

severe burns,

toxaemia,

prolonged illness,

multiple operations.

In my experience two such cases have responded to intravenous cortisone when all other measures have failed.

CONCLUSION

1. The introduction of cortisone and allied compounds in the past ten years has made possible adequate replacement therapy for major surgery on the adrenal and pituitary glands or in disease of these glands.
2. On the other hand, the extensive use of cortisone and allied drugs in high doses for many medical conditions has considerably increased the incidence of post-operative adrenal insufficiency.
3. Not every patient who has had adrenal suppression following cortisone will develop acute insufficiency with operative stress, but it is not always possible to foresee which type of patient this will be. Hence vigorous treatment with cortisone before, during and after operation is recommended.

The patient with adrenal suppression must be regarded as having the same instability as the diabetic patient in the presence of trauma, sepsis, haemorrhage or even severe pain and no delay in the administration of cortisone is permissible because of the rapid mode of death in this condition.

There is little doubt that high cortisone dosage to the surgical patient does result in the necessity for very careful post-operative management and that some complications, particularly sepsis and electrolyte imbalance, have a slightly higher incidence. These complications are of minor importance compared with the possibility of a fatal outcome in the post-operative stage, even in minor operations, from acute adreno-cortical insufficiency.

REFERENCES

FRANKSSON, C., GEMZELL, C. A. and VON EULER, U. S. (1954), *J. clin. Endocr.*, vol. 14 page 608.

GALANTE, M., RUKES, M., FORSHAM, P. H. and BELL, H. G. (1954), *Surg. Clin. N. Amer.*, vol. 34, page 1201.

HARNAGEL, E. E. and KRAMER, W. G. (1955), *J. Amer. med. Ass.*, vol. 149, page 1542.

HOWLAND, W. S., SCHWEIGER, O., BOYLAN, P. and DOTTO, A. C. (1956), *J. Amer. med. Ass.*, vol. 160, page 1271.

HUME, D. M. (1953), *Ann. Surg.*, vol. 138, page 548.

LATHAM, J. (1956), *Brit. J. Anaesth.*, vol. 28, page 77.

SALASSA, R. M., BENNETT, W. A., KEATING, F. R. and SPRAGUE, R. G. (1953), *J. Amer. med. Ass.*, vol. 152, page 1509.

TYLER, F. H., SANDBERG, A. A. and ECK-NES, K. 1952), *J. clin. Invest.*, vol. 32, page 608.

WILSON, P. (1956), *Med. J. Aust.*, vol. 1, page 289.

THE USE OF THE VEENEMA TROCAR IN PERINEAL PROSTATIC BIOPSY

By J. MADDERN

Adelaide

IN 1953, Veenema described a trocar for obtaining perineal prostatic biopsies. Following its use in 50 cases it is possible to give a clinical appreciation of the instrument in the diagnosis of cases suspected of having carcinoma of the prostate.

DIAGNOSIS OF CARCINOMA OF THE PROSTATE

Carcinoma of the prostate may present with symptoms due either to local obstruction or to metastases. The local obstruction may lead to difficulty of micturition, frequency, nocturia and occasionally haematuria. The metastatic lesions may cause "arthritis" and anaemia and occasionally thrombotic phenomena. These symptoms are vague and nonspecific but if the local obstructive symptoms have been present only a short time it may suggest carcinoma of the prostate. Clinically the diagnosis is suggested when on rectal examination an area of hardness or induration can be felt in the prostate or extending from it. Rectal examination is in fact the most valuable aid in the diagnosis of carcinoma of the prostate. The extent of the induration will vary from case to case. It may be confined to a nodule or more commonly a lobe or both lobes of the gland are hard. In the late cases there is extraglandular extension which can be felt extending laterally from the upper pole of the gland in the region of the vesicles. Distant metastases may be present in glands, liver or bone. In the latter case these are usually the classical osteosclerotic lesions. One may have difficulty differentiating these bone deposits from Paget's disease, especially if the gland is not unduly hard to rectal palpation.

Although rectal palpation is the most valuable aid in the diagnosis of carcinoma of the prostate it is not infallible. It has been shown that this clinical impression is attended with a considerable error (Colby, 1953; Kaufman *et alii*, 1954; Rinker and Shuman, 1952, and Semple, 1951). A similar clinical impression may be given by several conditions. In the case of nodules and lesions confined to a lobe the localized hard area may be due to a solitary prostatic calculus; less commonly it may be due to a benign capsular adenoma, an infarct or an inflammatory lesion. Jewett (1956) investigating the prostatic nodule has shown that excluding all cases which show a radiological calculus about half of the remainder are histologically carcinoma. In more extensive lesions benign hypertrophy, multiple prostatic calculi and inflammatory lesions may be misleading.

It is important in any malignant lesion to have histological or other irrefutable proof of the disease. Particularly is this so in carcinoma of the prostate whose clinical characteristics can be so altered by hormone therapy.

AIDS TO DIAGNOSIS

Hormone therapy has been given diagnostically in a doubtful case of carcinoma of the prostate. If the symptoms improve, or the gland shrinks in size, the diagnosis may be considered to have been confirmed. It must be remembered, however, that an inflammatory lesion may coincidentally melt away quickly during hormone therapy. While this is not due to the hormone itself, the hormone may be given credit. Such a test cannot be considered a scientifically accurate confirmation. Several other aids may be enlisted.

Besides the biopsy methods there is the serum acid phosphatase and also examination of the prostatic smear. In this latter method a specimen of prostatic secretion is obtained by prostatic massage and the smear is stained and examined for malignant cells. This is a difficult technique and requires a skilled pathologist to interpret the findings. Although there are some encouraging results reported (Frank, 1955; Peters, 1951), the general impression is that the method is disappointing (Colby, 1953; Goodwin, 1954).

The serum acid phosphatase, if raised, is a most valuable aid in the diagnosis of carcinoma of the prostate. It has, however, severe limitations. Woodard (1952) has shown that it will be elevated in 5 per cent. of cases in which the lesion is confined to the gland. In lesions which have locally invaded the soft tissues it will be raised in about 30 per cent. of cases. In lesions with distant gland and bone metastases an elevated reading is found in about 75 per cent. Biopsy methods give the most satisfactory confirmation of the clinical diagnosis. In the routine pathological examination of the prostate at post-mortem there are found many more cases of carcinoma of the prostate (Rich, 1935) than there are cases dying of the disease (Griswold, 1946). The significance of these cases of occult carcinoma is uncertain, however, if a gland is suspected clinically of being a carcinoma and the biopsy shows the histological characteristics of carcinoma no similar uncertainty exists, such a case must be regarded as carcinoma. A biopsy may be obtained by the transurethral, transrectal or perineal routes. Transrectal biopsy (Grabstald, 1955) carries with it a risk of infection and fistula and does not appeal. Transurethral biopsy is very satisfactory in advanced lesions but as carcinoma commonly begins in the posterior part of the gland, a transurethral biopsy unless extensive may miss the infiltrated area. The method also requires some skill and experience with the expensive and delicate instruments. Perineal biopsy is a method much more readily available and suitable for routine purposes. Open perineal biopsy which may be performed through the classical Young approach or by the subsphincteric approach of Belt has the advantage of allowing direct access to the suspicious area of the gland. It has the disadvantage of being an operation of some consequence and the troublesome complications of impotence, fistula and incontinence are not unknown. It is the most satisfactory method of diagnosis of small lesions in which only a nodule or a lobe are involved.

Trocar perineal biopsy which is to be described is a simple measure. It is easily performed and may be repeated if necessary. It causes little discomfort and is accompanied by minimal complications. The particular instrument used obtains pieces of tissue of adequate size to allow sections showing glands and stroma to be prepared. It has the disadvantage of all biopsy methods that a negative result may be of no significance. While there is some difficulty in obtaining biopsy material from a small area, with experience it is possible to direct the instrument with considerable precision. The instrument is very valuable in establishing the diagnosis in well defined clinical lesions.

THE VEENEMA BIOPSY TROCAR

(Distributed in Australia for V. Mueller and Co. by Drug Houses of Australia Ltd.)

The instrument was designed and described by Veenema (1953) (Fig. I). It has a sharp pointed end which is composed of two conical cups. These have cutting edges and cut the biopsy a little more than a 0.25 cm. in diameter. When approximated these constitute a fusiform end which allows easy palpation by a finger in the rectum and thus accurately guided into the gland or the suspicious area. The posterior cup is mounted on a movable shaft which is controlled by the finger and thumb.

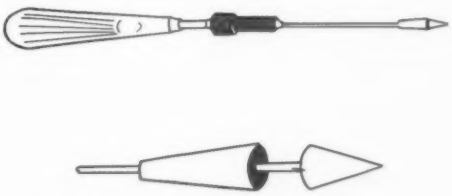


FIG. I. The Veenema Trocar.

Method of biopsy

The patient is given a general anaesthetic following a perineal shave and placed in the lithotomy position. It is convenient to cystoscope the patient first and then proceed to biopsy. A small skin incision is made anterior to the anus and with the left index finger in the rectum the instrument is introduced. The point of introduction is quite close to the anus and after directing the instrument anteriorly to avoid the rectal reflection it is passed almost horizontally thereby missing the vascular tissues of the bulb. As the trocar nears the gland it is guided by the rectal finger. When it enters the gland a definite resistance is felt. When in the gland or suspicious area the cups are opened and the instrument advanced a little. The cups

are then closed and the instrument rotated to shear off any tags of tissue. It is then withdrawn. The biopsy material is removed from the cups with a pair of tissue forceps. The procedure is repeated and several pieces of tissue from various parts of the gland or suspicious area are obtained. Bleeding which always occurs stops quickly with digital pressure. The small incision is left open. It is possible that the trocar may enter the bladder or urethra and it is wise to drain the bladder for twenty-four to forty-eight hours with a Foley catheter. The patient is given a soluble sulphonamide for five days.

Preparation of specimen

While it is possible to obtain good frozen sections from these biopsy fragments, it is most important to be able to examine each piece of tissue histologically. Unless this is done there will be cases of carcinoma missed. It is therefore important to prepare paraffin sections and several sections may need to be stained to ensure that all pieces of tissue have been histologically examined.

Complications

This series has been marked by a minimum of complications. Haemorrhage is always present but soon stops with digital pressure. In some cases there has been some bruising visible in the perineum a day or two after the operation. In the present series it has caused no difficulty. Infection has not occurred in the puncture wound in any case. In three cases there has been some post-operative fever but this has settled quickly. In one elderly patient there was considerable mental confusion and abdominal distension after the procedure. Bladder and urethral perforation has caused some mild haematuria but no other difficulty. Rectal perforation, incontinence and impotence have not occurred. Tumour implantation is a possibility and in common with all biopsy methods this could occur. It has not been apparent in any case in this series. There have been many thousands of this and allied procedures done and only one case of tumour implanation in the needle track has been reported (Clarke *et alii*, 1953). Presumably the risk is minimal and of little significance in carcinoma of prostate, deposits of which will be controlled in most cases by oestrogens.

Results

Fifty cases of suspected carcinoma have had a perineal biopsy using the Veenema trocar. There have been 37 cases in which a report of carcinoma was returned and in 11 the report was benign. In two cases the tissue examined contained no gland tissue and these are cases of failed biopsy. These cases occurred early in the series and with experience it is felt that this will be an unusual result. In such a small series as this no conclusions can be drawn about the efficacy of this method in the diagnosis of carcinoma of the prostate. It can be said however, that it is a useful method of obtaining prostatic tissue for histological examination. It has been most useful in the following three groups of patients:

(a) Diagnosis of cases which have been clinically suggested to be carcinoma of prostate.

(b) In establishing the diagnosis of the hard nodule in the prostate.

(c) In establishing the diagnosis in cases with extensive osteosclerotic metastases in which the prostate was not unduly hard.

In one case, extensive osteolytic lesions was associated with a hard nodule in one lobe of the prostate. The biopsy in this case was benign and the bone lesion eventually proved to be due to multiple myeloma. The biopsy has been helpful in cases which have had oestrogen therapy and in which at a later date some doubt of the original diagnosis existed. In these cases the pathologist has been able to give a definite opinion on the original lesion. Two cases were being treated as carcinoma who had in fact benign lesions, one a urethral stricture and the other an inflammatory lesion. Two further cases had the original diagnosis of carcinoma confirmed. In one case a report of benign hyperplasia was returned but subsequent examination of the gland removed at operation showed a small area of carcinoma. While this may happen with this biopsy and is its greatest disadvantage it is much less likely to occur if all of the removed pieces of tissue obtained are individually examined. In one patient a hard area in the prostate was due to a deposit from a hypernephroma.

SUMMARY

As the digital diagnosis of carcinoma of the prostate is open to a significant error, oestrogen therapy or radical surgery should not be begun on this evidence alone. Some confirmatory evidence should be obtained.

Trocar perineal biopsy is a useful method of confirming the diagnosis in:

(a) Cases in which carcinoma is suspected clinically.

(b) Hard nodules in the prostate.

(c) Cases with extensive osteosclerotic lesions in which the prostate is not unduly hard.

The Veenema trocar gives a simple and effective method of performing this with a good degree of accuracy and a minimum of complications.

ACKNOWLEDGEMENTS

Thanks are extended to the surgeons of the Royal Adelaide Hospital who have allowed access to their cases and especially to Mr. J. Estcourt Hughes and Mr. L. J. Opit for repeated help.

REFERENCES

CAMPBELL, J. S., LEADBETTER, W. F. and CLARKE, B. E. (1953), *J. Urol. (Baltimore)*, vol. 70, page 937.

COLBY, F. H. (1953), *J. Urol (Baltimore)*, vol. 69, page 797.

FRANK, I. N.)1955), *J. Urol. (Baltimore)*, vol. 73, page 128.

GRABSTALD, H. (1955), *J. Urol. (Baltimore)*, vol. 74, page 211.

GRISWOLD, M. H. (1946), *Conn. med. J.*, vol 10, page 106.

JEWETT, H. J. (1956), *J. Amer. med. Ass.*, vol. 160, page 838.

KAUFMAN, J. J., ROSENTHAL, M. and GOODWIN, W. E. (1954), *J. Urol. (Baltimore)*, vol. 72, page 450.

PETERS, H. (1951), *J. Urol. (Baltimore)*, vol 66, page 770.

RICH, A. R. (1935), *J. Urol. (Baltimore)*, vol. 33, page 215.

RINKER, J. R. and SHUMAN, W. G. (1952), *J. Urol. (Baltimore)*, vol. 67, page 709.

SEMPLE, J. E. (1951), *Brit. med. J.*, vol. 1, page 1236.

VEENEMA, R. J. (1953), *J. Urol. (Baltimore)*, vol. 69, page 320.

WOODARD, H. Q. (1952), *Cancer*, vol. 5, page 236.

Royal Australasian College of Surgeons

Proceedings

The College in New Zealand

As has been frequently emphasized, one of the main objects of the College is to improve the facilities for surgical training in Australasia. In Australia, the College has in the last ten years improved facilities for candidates for the Primary Fellowship Examination by increasing the frequency of the Primary Examination, which is now held alternately in Melbourne and Sydney in March and September of each year. The Post-Graduate Federation in Medicine has undertaken to hold courses for the Primary Fellowship in Sydney and Melbourne and details of these courses are advertised in the *Medical Journal of Australia*. The College greatly appreciates this co-operation by the Post-Graduate Federation.

The Council of the College has now decided to offer the same assistance to candidates in New Zealand, by conducting the Primary Examination twice a year. Arrangements have been approved whereby the examination will be completed in three or four days, the papers and the *viva voce* sessions being held upon consecutive days; this should lessen the burdens of time and expense for candidates coming from Auckland to Dunedin for the examination. It is hoped that arrangements will soon be completed whereby the examination will alternate between Auckland and Dunedin. Training courses for the Primary Examination have been organised in Dunedin and the possibility of similar courses in Auckland is being investigated.

With regard to training for the Final Fellowship, the College has always insisted upon training by apprenticeship, in the Registrar type of post in a hospital. The Council is strongly of the opinion that applicants for these apprentice posts should already be in possession of a Primary Fellowship. It should be the aim of Fellows, both in the Dominion and in the Australian States, to use their influence to see that these posts are available to the candidates who pass the Primary. We urge these young men to take the Primary; if they do this, we owe it to them to see that posts are available to them in Australasia, posts which will train them adequately for their Final Fellowship.

Having brought them thus far, to the possession of a Fellowship, it is obvious that the College must go further and help to provide the post-examination training, for which there is no time limit. This will include hospital appointments as surgeons and the organization of regular scientific meetings. Of a necessity, this work throws a great burden upon local Committees. The Dominion Committee at present meets twice a year, in conjunction with scientific meetings. At the suggestion of the Dominion Committee, the Council has decided to ask the Dominion Committee to hold two additional meetings each year in Wellington, the travelling expenses of Committee members to these two extra meetings to be a charge upon the funds of the College. Council also decided that if the President be an Australian Fellow, he should visit New Zealand once during his term of office. The President has accepted an invitation to attend the Annual Meeting of New Zealand Fellows in Dunedin in March, 1960. An invitation to all Australian Fellows has been published in the *Medical Journal of Australia*.

General Scientific Meeting, Adelaide, August,1960

Professor J. C. Goligher of Leeds and Mr. Norman Tanner of London will be in Australia and have been invited to attend the Meeting and contribute to the scientific programme.

A Business Meeting of Fellows will be held on 21st August, during the above meeting. Any Fellow who wishes to introduce matter for discussion is requested to give due notice of motion to the Secretary of the College not later than 30th June, so that an adequate Agenda Paper can be sent to Fellows, as was requested at the Business Meeting held in Sydney, 19th August, 1959.

Articles of Association

The Council has had the Articles of Association under review; and on the advice of the Solicitors of the College, certain amendments have been proposed. Subject to the approval of the Attorney-General of Victoria, these amendments will be submitted to a special General Meeting of Fellows for their consideration and approval. It is hoped that this meeting may be held in February, 1960.

National Register of Approved Surgeons

It has long been the objective of the Council to seek the acceptance of the Diploma of F.R.A.C.S. as the standard of the trained surgeon. To this end, the Council has decided to establish a National Register of Approved Surgeons. Such a register will be of value in the matter of hospital and teaching appointments; and in the matter of teaching the public that surgeons are trained, not born. If ever the political need should arise for a Register of Surgeons, it is desirable that the College should already have the matter in hand. The Council prefers the term "approved" surgeon, rather than that nebulous title "specialist."

Primary Fellowship Examination — Melbourne, Sydney and Dunedin, September, 1959

The following candidates satisfied the Board of Examiners at the September, 1959, Primary Examination for Fellowship held in Melbourne, Sydney and Dunedin, and were approved:—

Brian Maurice Barrett, Alistair Duncan Bird, Timothy Boyd Cartmill, Leslie James Caust, John Morton Copeland, Thomas Parker Davis, William Napier Etheridge, John Mackenzie Ham, Irwin Hunter Hanan, David Frederick Hogg, Thomas Benedict Hugh, Gabriel Andrew Kune, Donald Roger Marshall, Peter John Millroy, John Bentham Morris, Kenneth Arthur Myers, Ian Alexander Penn, Alan William Searle, James McKinnon Watts.

The Gordon Taylor Prize was shared equally by David Frederick Hogg and Ian Alexander Penn.

Primary Fellowship Examination, Faculty of Anaesthetists — Melbourne, Sydney and Dunedin, September, 1959

The following candidates satisfied the Board of Examiners at the September, 1959, Primary Examination for Fellowship of the Faculty of Anaesthetists, held in Melbourne, Sydney and Dunedin, and were approved:—

Michael John Bookallil, Harold Wah Kim Chan, James Ivor Clayton, Nancye Eunice Edwards, Evan Charles Hallett, Robert Mercer Hart, James Loughman, Donald Charles Maxwell, Newton Potter, Bryan Edmund Sharkey.

The Renton Prize was shared equally by James Loughman and Bryan Edmund Sharkey.

Final Fellowship Examination — October, 1959

A Final Examination for Fellowship of the College was held in General Surgery and all special branches of surgery in Melbourne in October, 1959. Sixty-eight candidates presented for the examination, of whom forty-eight were approved by the Court of Examiners. The names of successful candidates will be submitted to the Council at the next meeting and on admission to Fellowship will be published.

Faculty of Anaesthetists Final Fellowship Examination — October, 1959

At the Final Examination for Fellowship of the Faculty of Anaesthetists held in October, 1959, six candidates presented and all were approved. Their names will be submitted to the Board of the Faculty and to the Council of the College and on admission to Fellowship of the Faculty they will be published.

LEONARD LINDON,
President.

Books Reviewed

TREATMENT OF CANCER AND ALLIED DISEASES.

Vol. 1. PRINCIPLES OF TREATMENT.

Edited by GEORGE T. PACK, M.D., F.A.C.S., and IRVING ARIEL, M.D., F.A.C.S. Second Edition. New York, U.S.A.: Paul B. Hoeber, Inc., 1959. 10" x 7", xiv plus 646 pp., 505 illustrations. Price: $22.50.

This nine volume work edited by two famous surgeons, George Pack and Irving Ariel, represents the second edition to which 55 authors will contribute. The production of the first two volumes is excellent and the illustrations clear. The subject matter is authoritative, detailed and remarkably comprehensive. Every phase of practical oncology will be covered. Surgeons will hasten to congratulate the authors upon the importance and authenticity of their undertaking and its contribution to modern cancer therapy.

The philosophy of the first volume is based upon the contention that there is little difference between the management of cancer and that of other incurable diseases (diabetes, chronic nephritis, etc.). Not every Australasian surgeon will accept this and certainly very few nurses and laymen in the antipodes. This attitude is in many cases commendable in that it uses every modern surgical ancillary to support a courageous and aggressive attack on cancer. Every surgeon is aware that such an approach is far better than timid and ineffectual surgery but the philosophy remains questionable.

There is authoritative information on industrial cancer, cancer detection, cancer records and the home care of cancer patients. Diagnostic pathology is very well done and this is not to be wondered at as the chapters are written by Broders, Papanicolau and Stout.

The preparation of the patient for anaesthesia and operation, and the post-operative management is in line with modern thought in resuscitation and electrolyte therapy. A detailed chapter on vascular surgery and its application to operations for cancer is a new departure which will appeal to surgeons, although "haemostasis" may appear elementary.

Chemotherapy and hormone therapy adequately summarize what a clinician should know of these methods of therapy but the large section on radiotherapy is not so satisfying. Radiotherapists will not find a proper review of the role of pre- and post-operative radiotherapy or to the mutagenic and carcinogenic effects of radiation. Many pages of depth dose data are of no surgical interest and are irrelevant to radiotherapists who would have their individual data for their machines. The cause of this defect is an attempt by many authors to reach all members of the oncological audience simultaneously — a very difficult task. It might prove worthy of future consideration to put forward basic principles of radiotherapy for the surgical reader here and deal with indications and methods in the regional volumes later.

This volume is a worthy introduction to a great modern clinical compendium on oncology.

LECTURE NOTES ON MIDWIFERY.

By T. F. REDMAN. Bristol, England: John Wright & Sons Ltd., 1958. 7¼" x 4¾", 212 pp. Price: 12s. 6d. (stg.).

This book written in lecture note form, is in some respects too advanced for the average obstetrical nursing trainee and emphasizes the theoretical at the expense of the practical side. For example the chapter on normal labour hardly mentions the detailed steps of a normal delivery which is probably one of the most important tasks a midwife has to perform. No reference exists to the all-important act of properly naming the baby. On the other hand the chapter on infection lists the various components of the Gram stain, and refers to the morphology and staining characteristics of some common bacteria. A nurse should be able to advise a patient on certain aspects of ante-natal care, particularly diet. The section on diet does not elaborate sufficiently and certainly a patient could not understand it.

The basis of any teaching is the principles of a subject, yet these are rarely referred to nor stressed. This deficiency is most obvious with post-partum haemorrhage, one could imagine a sister being suddenly confronted with such an emergency, and not being able to remember what to do because she did not understand nor know the principles of treatment involved.

It must be remembered that this book was written primarily for a country where domiciliary obstetrics is common, and therefore the midwife has to perform more duties than in a place like Australia. Admitting all this, surely such procedures as manual removal of the placenta, especially without an anaesthetic is not a task for a nurse. One might deduce that per vaginam examinations by a midwife were common place. Other points of criticism are ante-natal attendances, the first within twelve weeks, and the second not until twenty-four weeks, no oxygen nor sucker in the room for eclamptic patients, the irregular use of ergomtrine in a normal delivery and the use of a "drip" into the inferior vena cava using a polythene catheter through the internal saphenous vein, rather than the normal simple "drip" into a vein in the forearm in the treatment of anuria.

SURGERY OF THE STOMACH AND DUODENUM.

By CLAUDE E. WELCH. Third Edition. Chicago: Year Book Publishers, Inc., 1959. 8½" x 5¾", 405 83 plates. Price: £5 7s. 3d. (Aust.).

This is not a good book. What "handbook of surgery" is? The volume under review aims at instruction in the operative surgery of the stomach and duodenum and it really falls a long way short of what seems to be the author's purpose.

The Billroth I procedure which, when correctly performed, can be one of the most elegant interabdominal operations, is mutilated beyond recognition, both by the text and the illustrations. Indeed all through the book the illustrations, which do not even have the virtue of being diagrammatic, are coarse and unsatisfying.

It would be idle to quote at length further examples of this volume's shortcomings, but to make some points perhaps the following may suffice: In discussing the management of haemorrhage complicating peptic ulcer, the author writes—

> "Ideally the operative procedure should secure complete haemostasis and render a cure of the underlying lesion. In other words, gastric resection including removal of the ulcer is best. This occasionally may have to be modified by circumstances."

What profound surgical proposals! Then wedge resection of gastric ulcer, pages on five or six different ways of performing gastrostomy and other obsolete operations are discussed.

Finally, I part company with the author on page 262 on the points of arterial ligation. One of the figures is rather frightening to someone like myself who has ligated quite a few left gastric arteries. On this matter the author states—

> "At the risk of introducing material that may insult the reader's intelligence, word must be said about ligation of the splenic left gastric and other large arteries."

When it is thereafter recommended that such vessels be crushed with three clamps and ligated with No. 1 chromic catgut, the reader's intelligence is indeed insulted and it is high time to part company with the author.

FRACTURE SURGERY — A TEXTBOOK OF COMMON FRACTURES.

By HENRY MILCH, M.D., F.A.C.S. New York: Paul B. Hoeber, Inc., 1959. 10¼" x 7", x plus 470 pp., 480 illustrations. Price: $17.50.

The senior author is consulting orthopaedic surgeon to the Hospital for Joint Diseases, New York, and has published many papers, the other is a resident surgeon at the Peter Brent Brigham Hospital, Boston. Their object was to design a book that filled the gap between the small handbook and the encyclopaedic specialistic volume. This book is meant for the medical student, the surgical house officer "as well as for the general practitioner . . ." A considerable space is devoted to large, excellent and numerous illustrations and radiographs which show a tendency to demonstrate the individualized views, experiences in rare and important complications and the author's general technique of fracture treatment. At times this treatment is of a technical and specialistic nature that tends to be rather above that usually practised in this country by the audience for whom the book has been written. For example, the technique of intramedullary nailing for fractures of the clavicle is heavily illustrated by one and a half pages although "this should not be used routinely." The many illustrations show the use of skeletal traction by various methods including even the notorious os calcis tongs, all sorts of medullary nails, bone grafts cut with an electric saw, repairs of arteries, nerves and tendons and other advanced procedures. In the section on anaesthesia we find the stellate ganglion block and an illustration of nerve blocking for open reduction and nailing of the femoral neck in elderly and debilitated patients. When we come to fractures that might be treated simply such as metacarpals, illustrations include pulp traction with the finger straight (anathema to Bunnell) operation and intramedullary fixation when angulation is more than 15° and an elaborate apparatus called the Goldberg splint which appears to correct the posterior angulation by screws and clamps and felt padding held in a plastic frame which is incorporated in the plaster forearm splint. Baby Thomas splints that hold the fingers extended are recommended for phalangeal fractures. In the lower extremity again there is the tendency at times to illustrate rare maladies such as injuries to the ischium (six large illustrations) which has been the subject of a recent monograph by the senior author, anterior transposition of the peroneal nerve and synostosis operation for persistent non-union of the tibia (9 illustrations). Dislocations are not included except the rare congenital recurrent dislocation of the elbow and "so-called dislocation of the lower end of the ulna" in both of which the author has a special interest as he has described operations for their cure in the past. The specialist may find interest in this book, but the resident medical officer and general practitioner will have to decide for themselves.

ORTHOPAEDIC SURGERY.

By Sir WALTER MERCER. Fifth Edition. London: Edward Arnold (Publishers) Ltd., 1959. 9" x 6½", xi plus 1,075 pp., 422 figures. Price: £4 10s. (stg.).

It is now more than a quarter of a century since the first edition of Mercer's "Orthopaedic Surgery" was published in response to many requests that the teaching of the Edinburgh School in this surgical specialty should be available to a wide audience. It can surely take a place among the classics of British surgical literature. The fact that new editions have been regularly called for is a clear indication that it fulfils its purpose of providing a useful reference book for students, for post-graduates reading for a senior degree, or for the general practitioner who wishes for authoritative guidance on an orthopaedic subject. In a book of moderate size, by careful selection of subject matter and good journalism, a particularly comprehensive cover of general orthopaedics has been achieved.

The important subject of congenital dislocation of the hip is discussed in Chapter 2 and gives the student a comprehensive survey of current thought and practice. Shelf reconstruction of the acetabular roof still has a place in the late case, but one is surprised to see the advice that the restored margin should be held in place by "a cortical graft from the bone bank secured by two ivory pegs."

Generally the section on congenital deformities is well presented, but the treatment advocated for talipes equinovarus would not be acceptable to most surgeons who have to deal with this troublesome condition. In spite of Denis Browne's strong advocacy many would criticize the statement that "the important element of the deformity is the longitudinal bending of the forefoot. If this is completely corrected the calcaneal deformity is automatically improved and the equinus also tends to disappear." Treatment along these lines all too often results in convexity of the sole.

Smillie's advice is quoted "that internal derangement of the knee in a woman should be regarded as recurrent dislocation of the patella until proved

otherwise." The author practices complete transference of the infra patellar ligament and its attached segment of bone to the medial side of the tibia and has found this operation entirely satisfactory. The fact that most cases of recurrent dislocation in the adult are associated with damage to articular cartilage and patello-femoral osteoarthritis is not mentioned, nor is patellectomy considered worthy of mention.

These criticisms however are of small consequence in what is a valuable treatize on current orthopaedic practice. All chapters are well set out with brief discussions of pathology and anatomy and a comprehensive survey of diagnosis and treatment. In the chapter on low back pain, it is pleasing to find that emphasis is placed on a careful history and painstaking examination.

This is a book which can be thoroughly recommended to the final year student, the post-graduate with surgical aspirations, and for the general practitioner who has not time to peruse nor access to specialist journals.

THE SELLA IN HEALTH AND DISEASE.

(Supplement No. 8 in British Journal of Radiology.)

By MAHMOUD EL SAYED MAHMOUD. London: British Institute of Radiology, 1958. 9¾" x 7¼", xii plus 100 pp., several illustrations. Price: 35s. (stg.).

This is a thesis which was submitted to London University for the degree of Ph.D. It is, in the words of the author, "an attempt to assess the value of the radiographic changes in the sella turcica caused by intracranial tumours and also an endeavour to correlate these changes with the site and nature of the tumours. Further, it attempts to correlate the histological changes of the sella turcica with its microradiographic picture and to correlate both with the conventional radiographs."

In the opinion of the reviewer the author has succeeded in his stated purpose. He has reviewed adequately the normal anatomy of the sella, from a material of 100 normal dry skulls, 100 normal autopsy cases and 25 normal, formalin-fixed brains for details of the third ventricle. The radiological anatomy has been reviewed from a study of 2,000 lateral skull radiographs. All of the radiographs were taken at the National Hospital, Queen Square, London, using a Lysholm skull table with a focus film distance of 28in. Rather than measure the size of the sella turcica, the surface area has been measured in 100 normal cases. Employing this technique the surface area varied between 22mm² and 130mm², but 50 per cent. of the measurements were between 60 and 100mm². The details of the thickness, length and shape of the dorsum sellae and of the floor of the sella are described in detail. This represents one of the most valuable sections of the book. In addition, in 19 patients the sella turcica has been studied histologically and microradiographically. This study explains the normal radiographic appearance of the sella in lateral projection and has been compared with a further study of 17 patients with verified intracranial tumours, 8 of whom showed abnormal and 9 showed normal radiological appearances of the sella.

These studies demonstrate that radiological changes observed are due to patchy bone destruction, new bone formation and changes in the subcortical spongy bone. There is no histological or microradiographic evidence of decalcification, only of destruction of bone. The author points out that it is incorrect to speak of "decalcification of the sella" in a radiological report.

The remainder of the book is concerned with a discussion on the radiological changes in the sella in a total of 615 verified, intracranial space occupying lesions. Separate studies were made of changes present in tumours of the sella walls, intrasellar tumours and cerebral tumours situated above, in front of, lateral to and behind the sella.

A separate study was made of the tumour material according to pathological classification; further studies were made of the effect of vascular hypertension on the sella and of the relationship between the degree of pneumatization of the sphenoid sinuses and the presence of pathological changes in the floor of the sella in cases of intracranial tumour.

Among the new material there were 49 cases of proven intrasellar pituitary adenomata. It is of considerable interest that in only four of these the area of the sella was less than 130mm² and in the remaining 45 the area of the sella was above 130mm², the maximum being 823mm². All cases with normal sized sella were of the eosinophilic type and showed clinical and radiological evidence of acromegaly. It would therefore appear that the author's method of relying on surface area of the sella as shown in radiographs taken under standard conditions is an extremely reliable one of detecting intrasellar tumours.

Several minor criticisms may be made about this work. As with most published theses, it has been necessary to group the illustrations on art paper to reduce the cost of the publication. Also it is apparent in several places that the author is not writing in his native tongue. However, in spite of these minor points, this is a comprehensive study of the sella which should be studied by all those interested in the neurological sciences and endocrinology and particularly by radiologists.

THE MAST CELLS.

By J. F. RILEY. Edinburgh and London: E. & S. Livingstone Ltd., 1959. 10" x 6½", xvii plus 182 pp., 65 illustrations. Price: 30s. (stg.).

When Ehrlich gave his paper on mast cells to the Physiological Society of Berlin in 1879, he stressed the plumpness of these cells. He noted that they were abundantly present in hypertrophic connective tissue such as young keloidal tissue and in the tissues affected by lymphoedema. Consequently he thought they were concerned with the nutrition of connective tissue. In 1937 Swedish workers produced fairly good evidence that the metachromasia of mast cells is due to the presence of heparin. The next advance in the story of the mast cells was in 1953 when James F. Riley found that histamine-liberators reacted with mast cells. Together with G. B. West he has shown that mast cells contain just as much histamine as heparin and the concentration of the substances parallels the density of the mast cell population. In urticaria pigmentosa and mast cell tumours very large amounts of these substances are present.

Strangely enough, in the anaphylactoid response to histamine-liberators, the dog is the only animal in which there occurs an increase in heparin in the

blood, and an increase in blood-clotting time. In other animals histamine-liberators disrupt mast cells but heparin does not enter the blood stream in detectable amounts. These facts have led Riley back to the original hypothesis of Ehrlich, that the chief function of mast cells is in the growth and maintenance of connective tissues and that heparin may be a precursor of hyaluronic acid.

In this monograph Riley presents a review of the literature up to 1950 and thereafter the book is mainly concerned with experimental histamine-release studies of the author.

Although the function of mast cells is obscure they undoubtedly play some part in inflammation and anaphylaxis. Therefore anybody interested in these biological phenomena should acquire this book which contains almost all the known facts about mast cells and is both well written and well illustrated.

OUTLINE OF ORTHOPAEDICS.

By JOHN CRAWFORD ADAMS, M.D., F.R.C.S. Second Edition. Edinburgh: E. & S. Livingstone Ltd., 1958. 8½" x 5½", vii plus 428 pp., 301 figures. Price: 35s. (stg.).

This short book covers the larger part of the subject of orthopaedics most adequately. Since it is written on a regional basis, it will be of great help to the undergraduate and the general practitioner, as a manual of orthopaedic differential diagnosis. Fractures are not included, and hence the subjects are able to be dealt with briefly and adequately.

The author has not forced his own views on treatment but has given a concise description of the current methods on controversial aspects.

Mr. Crawford Adams is to be congratulated on this production.

LUMBAR DISC LESIONS, PATHOGENESIS AND TREATMENT OF LOW BACK PAIN AND SCIATICA.

By J. R. ARMSTRONG, M.D., M.Ch., F.R.C.S. Second Edition. Edinburgh: E. & S. Livingstone Ltd., 1958. 10" x 6¾", xii plus 244 pp., 21 plates, 60 figures. Price: 45s. (stg.).

This book supplies a most adequate coverage of the present state of knowledge of the causes of low back pain. Whilst Mr. Armstrong may shock many, by his contention that the old-fashioned lumbago or fibrositis is related to the pathology in the discs, there are many facts to support it.

Low back pain, the *bete noir* of the orthopaedist and most other practitioners, is clearly explained in its various aspects, and in most instances, ascribed to disc degeneration and prolapse.

The author's claim that 10.7 per cent. of operative cases have multiple disc prolapses, will come as a surprise to many. Do we not look far enough at operation, or what are the criteria of prolapse?

The treatment of the disc syndrome by spinal grafting is dealt with in what appears to be the right perspective at present; that is, the great incidence of failure of fusion is emphasized.

It is amusing to consider, that due to the hydrostatic balance of the discs, without the compressing force of gravity, we become longer in bed.

This is a most lucid book, and is commended to all who bear the cross of treating low back pain.

SURGERY IN WORLD WAR II — NEUROSURGERY.

Edited by Drs. R. GLEN SPURLING and BARNES WOODHALL. U.S.A.: U.S. Government Printing Office. 10" x 6¾", xix plus 465 pp., 130 illustrations, 19 tables. Price: $5.00.

This volume consists of two parts. The first is concerned with administrative considerations and in retrospect it is the problems of administration which appear the most interesting for it indicates the growth of an organization which in 1942 existed only in the minds of a few and then perhaps in a rather sketchy fashion to one spread over a great portion of the globe. The problems encountered in the enlistment, training and placing of the personnel required are described.

For some time after the entry of the U.S.A. into World War II neurosurgical work was little different to that experienced by neurosurgeons in peace time. However, difficulties were soon experienced when some of these problems were encountered against a background of service life. This experience led to the early discharge from the army of patients with intervertebral disc lesions before any active treatment could be instituted, while peripheral nerve lesions were given a "work furlough" for long periods, so that they could be more profitably employed in civilian occupations.

Throughout this early period information was disseminated by conferences and a monthly newsletter circulated to all neurosurgeons in the service, but as so often happens in wartime there was little attempt to rotate surgeons between forward and rear units, a far superior method of disseminating information and experience. Gradually the organization increased in size but it would appear that the medical staffing of most units was too small to be efficient. The shortage of ward medical officers was particularly acute. One neurosurgical centre with 1,450 patients had a total staff of eight medical officers. Such a situation could only lead to overwork of the medical staff and stagnation of the patients, while often the trained and experienced neurosurgeon was bogged down with administration while his assistant neurosurgeon did the operating, probably taking twice the time and doing it only half as well.

By the end of 1945 the zone of the interior had 21,900 neurosurgical beds but at this time there was only one neurosurgeon to every 237 patients.

To cope with the difficulties the army introduced courses of training, but at first the candidates were few and the difficulties were constant. It was finally concluded that "experience in the training programme supported the belief that in any future war, the procurement of trained neurosurgeons must depend primarily upon a pool of existing talent, supplemented by well-grounded general surgeons who are taught intensively the well-defined types of neurosurgical injuries which form a common pattern in warfare."

Opinions concerning the organization of neurosurgical units while on active service differed in the various theatres of war. In the Mediterranean theatre neurosurgical teams were placed in forward hospitals 15 to 50 miles behind the front. While a larger neurosurgical centre was placed amongst a group of general hospitals in the base area. In the European theatre, an attempt was made to place a

neurosurgical team in each evacuation hospital but trained personnel were too few and the standard of work in some centres was inadequate. Moreover there was a general wastage of trained staff. During the whole of the campaign, from Normandy to Berlin, the average number of cases handled by each neurosurgeon was only 150; with greater mobility of teams less men could have achieved much more. It is of interest that the final scheme proposed for this situation was almost exactly similar to that developed by Sir Hugh Cairns for the British Army in 1940.

The second and larger portion of the book consists of monographs on the management of various types of cranio-cerebral injuries encountered in wartime. Much of this information has already been published and is now regarded as standard procedure. Nevertheless the chapter on acute cranio-cerebral injuries written by Matson is certainly worth recording once again, for it outlines the principles of neurosurgical procedures which a younger generation of general surgeons or even some neurosurgeons would do well to read. Another interesting chapter concerns a clinicopathological analysis of 22 cases of fatal penetrating brain wounds.

The appendices cover a wide range of information, but the section on management of neurosurgical emergencies is particularly well set out. While the special history and examination forms which appear in great detail are worthy of study by those concerned in the assessment of the end results of head injuries.

GLAUCOMA — TRANSACTIONS OF THE THIRD CONFERENCE, January 8, 9, 10, 1958.

Edited by F. W. NEWELL. New York: Josiah Macy Jnr. Foundation. 9" x 6", v plus 272 pp. Price: $5.25.

During the past fifteen years the Josiah Macy Jr. Foundation has organized more than 20 conference groups. Each group has met for at least two days annually over a period of five or more years. Each meeting is limited to 25 participants selected to represent a wide approach to some urgent problem. Speakers present the most interesting aspects of his research in an informal manner, rather than in the form of a paper. Interruptions in the form of questions or comments are encouraged.

The group interchange in these conferences has been proved itself to be an effective way of improving understanding, broadening perspectives, changing attitudes and removing prejudices. As an experiment this method has shown the "the major obstruction to understanding among scientists lies in the resistance of human attitudes to change, rather than in difficulties of technical comprehension."

This volume contains reports of four discussions. The first was on tonography, and was opened by Morton Grant. Various methods of estimating the facility of outflow of the aqueous are described. He considered that neither measurement of intra-ocular pressure or of facility of outflow alone is a sure basis for separating normal from glaucomatous eyes. Frequent measurements of intra-ocular pressure during the day and at night on different days were considered as valuable as a measurement of facility of outflow. The existence of field defects in the presence of normal nerve heads was considered to be extremely rare, if indeed it ever occurs. Cupping of the discs however does occur without field defects. Baring of the blind spot is significant only when the upper or lower poles of the disc is affected. A facility of outflow of less than 0.20 is unlikely to be normal, whereas one that is greater than this is unlikely to occur in a glaucomatous eye.

The great clinical value of tonography is that it alerts the oculist to those patients who require careful follow-up. There are many patients whose optic discs withstand intra-ocular pressure of 30-35 mm. of mercury without loss of function. As we cannot estimate the vulnerability of the optic disc in such a patient we must rely on repeated tonometry or tonography.

The effects of application of a tonometer to the eye and of breathing and of blood pressure on measurements are discussed in detail. In healthy young adults a low coefficient of outflow may be due to a compensatory low rate of production occurring during a low period in the diurnal cycle. The necessity of waiting until the patient, especially if young relaxes is stressed.

The significance of the following histological findings is discussed: the minute holes described by Ashton and Dvorak-Theobald, endothelial cells with giant vacuoles and the mucopolysaccharide layer.

Variations in scleral regidity may affect the readings in tonometry. A low scleral rigidity can produce an apparent low facility of outflow. The new applanation tonometer followed by tonography permits correction of readings for scleral rigidity.

Glaucoma often first appears as a decreased outflow facility but with normal pressures — the cause being hyposecretion compensating for the decreased outflow. Probably many people go through life with a decreased outflow that is compensated by a secretory suppression.

A type of glaucoma due to hypersecretion is described in which raised pressure and field loss are associated with normal facility of outflow. Most patients were middle-aged females, and many had evidences of the "diencephalic syndrome."

A second discussion concerns the osmotic factors that take part in the formation of aqueous. It was opened by Barany of Sweden. Perkins of London introduced the third topic which was "Consensual changes in intra-ocular pressure under experimental conditions." After considering a central regulating mechanism and the influence of relaxation and tonometry itself, it was suggested that consensual lowering of pressure in the second eye was caused in part by passive lowering due to relaxation and in part by the oculocardiac reflex. The tonometer should not be regarded as a laboratory instrument. The average of 2-4 readings or the most frequent reading in a series is the one of importance. Readings with two weights are necessary if we are to exclude marked deviations from standard scleral rigidity. Much experimental work was described in this and the previous sections. The value of gonioscopy during operation was stressed as a guide to diagnosis and operation.

The clinical value of tonography was the last discussion. There was considerable support for the

value of tonography in evaluating the management and prognosis of glaucoma. It alone would not provide an indication for surgery. More correlation between reliable tonographic results and perimetry is essential. One clinic has already accumulated 25,000 tonographic tracings. Great diagnostic value was attached to the rates between the facility of outflow and the rise (8 mm. of mercury or more) in pressure after the water-drinking test.

A shift of emphasis is apparent from the effects of various techniques on the anterior portion of the eye to their effect on the posterior pole. It would be significant to know whether water-drinking and other tests induced scotomata or other evidence of field loss. The conference concluded with the statement that every patient from birth to death should be considered as a "glaucoma suspect."

SHAKESPEARE AND MEDICINE.

By R. R. SIMPSON, M.B., Ch.B., F.R.C.S., F.R.C.S.Ed. Edinburgh: E. and S. Livingstone Ltd., 1959. 8½" x 5½", vii plus 267 pp., 8 plates. Price: 25s. (stg.).

There is much of medical interest in Shakespeare's works. As the author points out in his introduction it is rarely appreciated how often Shakespeare uses the medical approach to illustrate a phrase or a situation; his references cover every aspect of medicine.

This book gives us a most excellent account of the medicine of Shakespeare introducing it to the reader in all its diverse categories.

One of the longest and most interesting chapters is devoted to John Hall, Shakespeare's medical son-in-law, with an excellent account of his life and work. Hall's manuscript case book, now in the British Museum, was translated and published by James Cooke in 1657. Hall must have been a good practitioner, in whom his patients had every confidence. It is suggested that Hall's character influenced Shakespeare's opinion of medicine and doctors in general in the plays published after 1600. Mr. Simpson points out that there were more medical allusions in Shakespeare's plays before Hall arrived in Stratford in 1600 than afterwards and that the number decreased markedly after he married Susanna in 1606.

All the well-known lines are here, together with many not so familiar, all delightful to read and re-read and giving in all a good picture of Elizabethan medicine.

This is the sort of book to have at one's bedside for quiet and frequent browsing. Much has been written on Shakespeare and medicine but Mr. Simpson must surely have prevented anyone else from taking up the subject in the future and, by so doing, has earned our thanks and congratulations.

ALFRED HOSPITAL CLINICAL REPORTS, Volume 8.

Melbourne: Brown Prior & Co., 1958. 10" x 6⅛", 108 pp.; illustrations.

This small volume contains ten papers written by members of the Medical Staff of the Alfred Hospital on diverse topics. That is, this volume lives up to its title and is a collection of reports.

Some of these papers are of real value, particularly the review of cases of haematemesis and melaena admitted over a nine-year period. This gives some interesting statistical information on this problem.

Dr. Luke's article on angiography of the aorta and main vessels, shows what strides are being achieved in diagnostic radiology. It is only one example of the work being carried out in the department of radiology. This volume cannot have a wide distribution since some of the papers are only case reports and all are brief. It is of value in showing the work being carried on in one of our major teaching hospitals. It is fitting that the first paper is a fascinating account of J. F. MacKeddie. There is no doubt that MacKeddie would have been delighted to know this work was being carried out and, more important still, published in such an attractive manner.

CANCER OF THE SKIN.

By J. C. BELISARIO. London: Butterworth & Co. (Publishers) Ltd., 1959. 8¾" x 5½", xvii plus 321 pp., including index, 14; 201 illustrations. Price: £3 8s. 6d. (stg.).

This outstanding monograph deals with the problems of diagnosis, management, and nomenclature of skin cancer. It comes at a time when some University lecturers and hospital teachers are disturbed at the extent of ignorance and bias shown by some teaching specialists, who adopt an ostrich-like attitude to any new approach outside of their particular field. This unhealthy outlook has caused considerable conflict and confusion in the minds of the younger dermatologists, surgeons and radiotherapists, who have had insufficient experience of the end results of treatment by techniques other than those used within their speciality.

Dr. Belisario has critically balanced the pros and cons of the previously accepted surgical and radiotheraupeutic approaches against those of chemosurgery, electrical, freezing and dermabrasion methods. His expert use of the combined cautery-curette-chemosurgical technique has produced such excellent results that it behoves all doctors — whether they refer their patients or treat them — to critically review their current habits of management This is especially important because of increasing public demand for perfection in treatment results.

The author is aware that some doctors may object to his new techniques of obtaining maximal cosmetic results on the ground that the recurrence rate may be higher than from routine radiotherapy or surgery. He has shown that recurrence is rarely a problem and in fact may best be treated with his methods. Those who object tend to forget that one of the rarest of all complications from basal cell carcinomata and early squamous cell carcinomata is metastasis and that we are not treating a killing disease but an anxious patient entrusting us with his cosmetic welfare.

One great value of this publication is its personal bibliography and copious references, which number over 500. There are over 200 excellent photographic illustrations, many of them before and after, and some in colour.

The book presents such startling contrasts to those which have been written by some radiotherapists, dermatologists and surgeons that it behoves all free-thinking members of the profession to consider its messages and to compare them with recent publications of a similar nature.